A. M. A.
ARCHIVES OF
OTOLARYNGOLOGY

EDITORIAL BOARD

GEORGE M. COATES, Chief Editor
1721 Pine Street, Philadelphia 3

JOHN D. KERNAN, New York
RALPH A. FENTON, Portland, Ore.
ERNEST M. SEYDELL, Wichita, Kan.
CHEVALIER JACKSON, Philadelphia
ROBERT L. GOODALE, Boston
J. MACKENZIE BROWN, Los Angeles
CARL H. McCASKEY, Indianapolis

RICHARD J. PLUNKETT, M.D., Chicago, Managing Editor

AUGUST 1951
VOLUME 54 NUMBER 2

Published Monthly by
AMERICAN MEDICAL ASSOCIATION
535 NORTH DEARBORN STREET • CHICAGO 10, ILLINOIS

Entered as Second Class Matter Jan. 14, 1925, at the Postoffice at Chicago, Under the Act of March 3, 1879. Annual Subscription, $12.00

When a Specified Schedule of Treatment is Important

NOW, THE DE VILBISS POCKET ATOMIZER MAKES NASAL MEDICATION POSSIBLE WHILE AT SCHOOL, BUSINESS OR TRAVELING

Every good prescription pharmacy carries the DeVilbiss Pocket Atomizer No. 33. Compact, convenient, leakproof, it enables you to prescribe a specified schedule of application for your patients. Regardless of the patients occupation or activity, you can control the continuity of treatment with the DeVilbiss No. 33. The DeVilbiss Company, Toledo 1, Ohio, and Windsor, Ontario.

De Vilbiss...

ATOMIZERS
NEBULIZERS
VAPORIZERS

The only broad-spectrum antibiotic available in concentrated drop-dose potency, Crystalline Terramycin Hydrochloride Oral Drops provide 200 mg. per cc.; 50 mg. in each 9 drops. Indicated in a wide range of infectious diseases, Terramycin Oral Drops are miscible with most foods, milk and fruit juices, affording optimal ease and simplicity in administration.

Supplied — *2.0 Gm. with 10 cc. of diluent, and calibrated dropper.*

ANTIBIOTIC DIVISION **Pfizer** CHAS. PFIZER & CO., INC., *Brooklyn 6, N. Y.*

Dependable Vacuum FOR OFFICE—BEDSIDE—SURGICAL USE

Handily portable, compact, always ready for instant use, this famous little Carmody aspirator develops 25 inches (Hg.) of vacuum—more than enough for most purposes—and ample (15 lbs.) spray pressure. It's a real convenience in the office or treatment room, and an ideal aspirator for carrying to home or hospital for bedside use.

The Carmody is a complete unit, equipped with a built-in vacuum gauge, control valve, filter in the spray stream and quart vacuum bottle. A standard suction tip, pressure cutoff and tubings are also furnished. The unit, on a lightweight cast base, with ventilated cast aluminum housing, measures only 6" x 14" x 11" overall.

The powerful little motor and rotary pump run quietly, without vibration. Automatic thermal overload protection avoids accidental burning out of the motor. Built with a minimum of moving parts, individually assembled, "run in" and bench tested, the Carmody unit will give you years of satisfactory, care-free service.

THE *Carmody* UNIT

Model AS-16

Complete, ready to operate on 110 volts, 60 cycles, alternating current, (f.o.b. Chicago) each, *$125.00*

Write for Your Copy of Our Complete New Brochure on Surgical Pumps

V. Mueller and Company 330 S. HONORE ST., CHICAGO 12, ILLINOIS

A safe, simple Mouth Gag for Tonsillectomy

Hazards of trauma pressure on the incisors by the usual mouth gag are eliminated by this simple instrument

CONTACT is placed behind the canine teeth. The instrument provides better exposure and fits any mouth...from small child to adult. Easily adjusted from side to side, always with two-point contact. Three sizes of tongue blades are provided. The contact portion of the ring is covered with small rubber tubing, as an added protection. This gag may be used on edentulous patients.

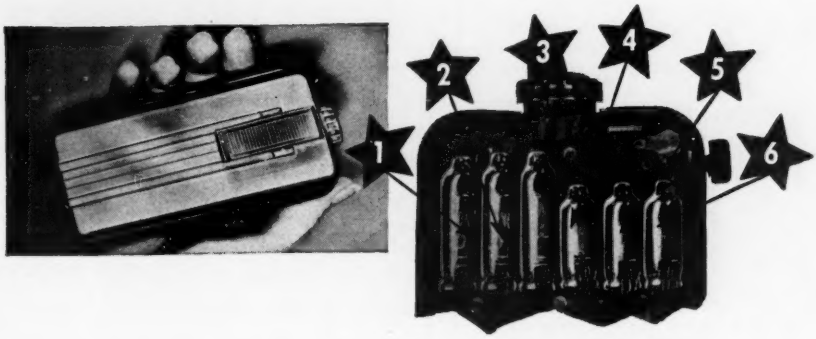
The new Atraumatic Mouth Gag, of fine material and workmanship, with three sizes of tongue blades . . . . COMPLETE **$25.00**

Consult your Dealer or write to...

THE ATRAUMATIC COMPANY 416 Thirtieth Street · OAKLAND 9, CALIFORNIA

NOW—the new 6-TUBE SONOTONE POWERHOUSE

brings hearing to the severely deafened



NOT 3 BUT 6-TUBE POWERED

Usable hearing—that is what the new Sonotone "966" will bring to three classes of your hard-of-hearing patients: (a) those whose losses are as high or higher than 80 decibels, who do not wear a hearing aid because none now existing can help them; (b) those hearing aid users whose deafness has progressed beyond the ability of their aids to help them; and (c) other users who are inadequately served by their present equipment.

Why is this true? Because the *6-tube* circuit provides *ten times the power* of the average hearing aid—the power needed for those whose residual hearing is so low that they had never hoped to hear again, power that reaches whatever useful hearing is left.

Powerful as the "966" is, the power is *controlled.* This new aid is readily adjusted to suit the individual. Extra loud sounds are modulated automatically. There is a tolerance control, an internal adjustment which gives a cut-off point beyond which further increase in volume is automatically limited. A bifocal control—adjusted by the wearer of the instrument—reduces background noise. The Sonotone external microphone banishes the annoying "sh-sh" of "clothes-rub."

The "966" is suitable for air and most particularly for bone conduction. Tell your severely deafened patients about this new way of coming back into the hearing world.

SONOFACT #5

Any amplifier will give *some* help to most hard-of-hearing patients, but a properly fitted Sonotone will make the *best* possible use of the residual hearing available. Sonotone does not utilize *one* instrument to fit all degrees of impairment but offers five for best possible fitting.

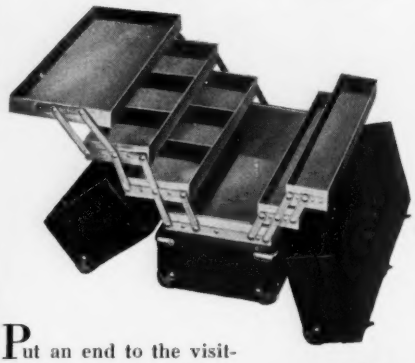
A request on your prescription blank will bring detailed information on the remarkably powerful Sonotone "966."

SONOTONE CORPORATION
Box A-81
Elmsford, N. Y.

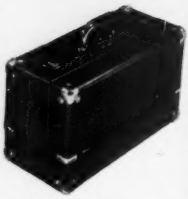
SURE CURE FOR "JUMBLE-OSIS"

(Disorderly medical bag)

NEW! **This compartmented medical kit keeps instruments neat, orderly, accessible.**



Put an end to the visiting doctor's greatest irritation — fumbling in a disorderly medical bag. This new, functionally designed kit, with many trays for small vials and a large section for large instruments is just what the doctor ordered.



A single glance tells you where each item is — everything in its place and a place for everything.

SPECIAL PURCHASE

MONEY BACK GUARANTEE **$24.50**

- **Extremely strong and durable**
- **Strong aluminum trays and lining**
- **Trays open and close telescopically**
- **Metal corners for added strength**
- **Sturdy frame and heavy fabric finish**
- **15½" long, 7" wide, 8¾" high**

MAIL THIS HANDY COUPON TODAY

Eagle Supply Co.
327 West 42 Street • New York, N.Y.

I am enclosing a check (or money order) for the new medical bag shown above, with satisfaction guaranteed or my money back.

Name.....

Address.....

City.....State.....

TO HELP YOUR PATIENTS AND SAVE YOU TIME

Booklets on Eyesight

These booklets prepared especially for the American Medical Association will answer many of your patients' questions for you. Among the titles, you will undoubtedly find topics you've had to discuss at length with patient after patient taking up time that you could have used to *treat* other patients. Let these booklets solve that problem for you.

WHOM SHALL I CONSULT ABOUT MY EYES? F. T. Jung. 8 pages. 15 cents.

EYES-RIGHT ! Audrey McKeever. 14 pages. 15 cents.

HOW TO SAVE YOUR EYES. Cleo Du Bois. 4 pages. 15 cents.

SHOULD YOUR CHILD WEAR GLASSES? J. Robert Burke. 12 pages. 15 cents.

NEW EYES FOR OLD CORNEAL TRANSPLANT. Max Fine. 10 pages. 20 cents.

GLAUCOMA. Arthur A. Knapp. 12 pages. 15 cents.

CATARACT. Abram B. Bruner. 12 pages. 15 cents.

CONTACT LENSES. Marguerite Shields. 3 pages. 10 cents.

COLOR BLINDNESS. Dean Farnsworth and Conrad Berens. 2 pages. 15 cents.

VISUAL EFFICIENCY. Henry A. Imus. 8 pages. 15 cents.

WHAT TO DO FOR BLIND CHILDREN. Park Lewis. 26 pages. 15 cents.

EYE PHYSICIANS, OPTICIANS AND THEIR WORK. Edward Jackson. 22 pages. 15 cents.

PERFECT EYESIGHT WITHOUT GLASSES. Meyer Wiener. 2 pages. 10 cents.

ONE EYE SEES EVERYTHING. Phyllis Duke-Elder. 2 pages. 10 cents.

Keep a supply on hand. Available at lower cost for quantity purchases. Write for prices.

AMERICAN MEDICAL ASSOCIATION
535 North Dearborn Street
Chicago 10, Illinois

In acute and chronic

Sinusitis

By using the NICHOLS NASAL SYPHON, these cavities may be safely and successfully drained of mucopurulent material, thus affording prompt symptomatic relief. Used and prescribed for more than 32 years. Prove its effectiveness on your most troublesome cases. Just mail the coupon.

Nichols NASAL SYPHON

NICHOLS NASAL SYPHON, INC. MONTCLAIR, N. J.

Please send details of SPECIAL OFFER "S" without obligation.

NAME..... M.D.

ADDRESS.....

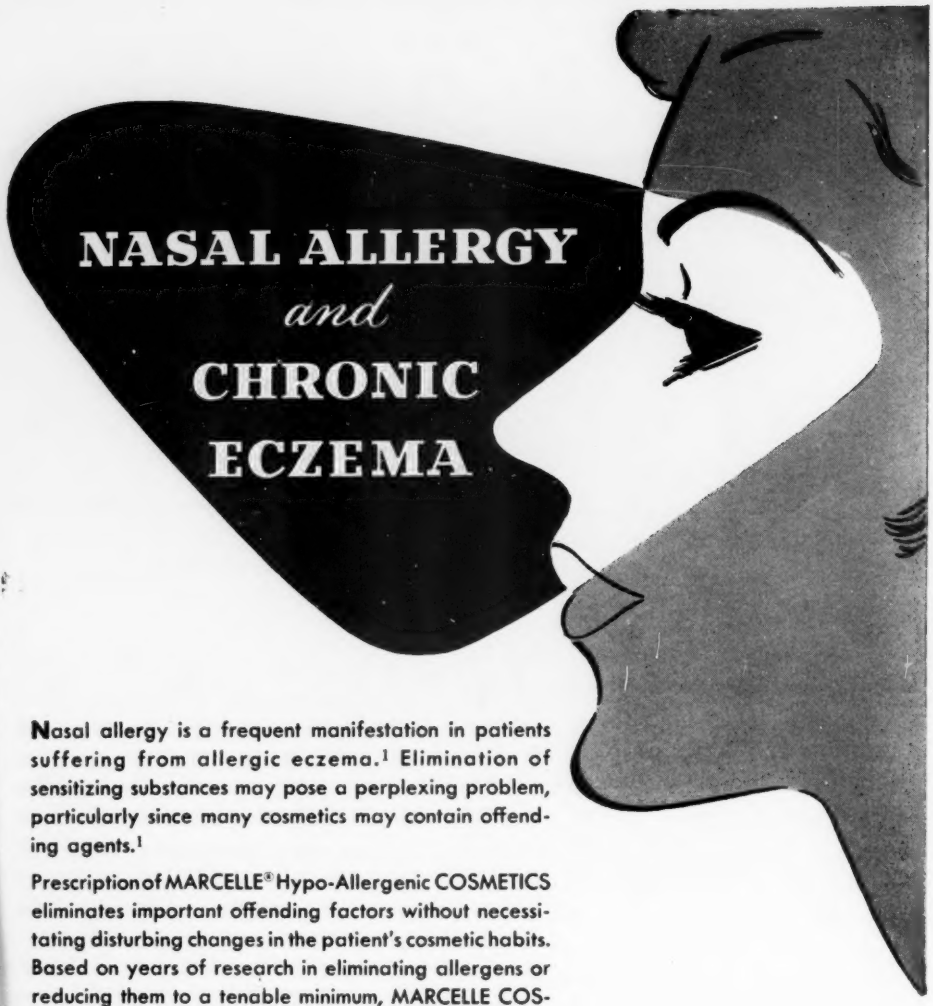
CITY..... ZONE..... STATE.....

The **SMR Suction** has no moving parts, belts to break, valves to wear out, or motors to burn out. Once installed there is no cost of maintenance. Some local installations have been in constant use for over 20 years without attention other than changing the rubber tubing. If necessary, the secretions can be trapped, but in practice the secretions are drawn directly into the sewer. The **SMR SUCTION** can be attached to any lavatory which has an exposed trap and can be removed when changing offices. Complete with vacuum breaker, valve, tubing, trap tee and gauge (calibrated in mm. Hg.). Should have at least 20 pounds of sustained water pressure. Price $32.50 f.o.b. Los Angeles.

SMR

Surgical Mechanical Research

1901-1903 Beverly Blvd. Los Angeles 4, Calif.



NASAL ALLERGY *and* CHRONIC ECZEMA

Nasal allergy is a frequent manifestation in patients suffering from allergic eczema.[1] Elimination of sensitizing substances may pose a perplexing problem, particularly since many cosmetics may contain offending agents.[1]

Prescription of MARCELLE® Hypo-Allergenic COSMETICS eliminates important offending factors without necessitating disturbing changes in the patient's cosmetic habits. Based on years of research in eliminating allergens or reducing them to a tenable minimum, MARCELLE COSMETICS are valuable aids in the prevention and management of allergic manifestations.

MARCELLE COSMETICS are the original hypo-allergenic cosmetics and the first line of cosmetics accepted by the Committee on Cosmetics of the American Medical Association.

1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

MARCELLE COSMETICS, INC.

1741 North Western Avenue, Dept. O • Chicago 47, Illinois

Marcelle
HYPO-ALLERGENIC
COSMETICS

ACCEPTED COMMITTEE ON COSMETICS AMERICAN MEDICAL ASSOCIATION

SAFE COSMETICS FOR SENSITIVE AND ALLERGIC SKINS

CONTENTS

PAGE

Malformations of the Auricle and the External Auditory Meatus: A Critical Review. Franz Altmann, M.D., New York 115

"Postoperative" Hyperrhinolalia (Rinolalia Aperta). Emil Froeschels, M.D., New York 140

Tracheotomy in Tetanus. Emanuel Herzon, M.D.; Edgar Killian, M.D., and Samuel J. Pearlman, M.D., Chicago 143

Treatment of Hay Fever by Hyposensitization. Solomon Slepian, M.D., Brooklyn 157

Neurofibrosarcoma of the Facial Nerve Involving the Tympanomastoid. M. Reese Guttman, M.D., and Marshall U. Simon, M.D., Chicago 162

Use of a Tantalum Foil Tube to Maintain Patency of the Nasofrontal Duct. William C. Hugenberg, M.D., and Henry L. Williams, M.D., Rochester, Minn.. 167

CASE REPORTS:

Congenital Neurogenic Malformation of the Faucial Tonsil. G. Kenneth Lewis, M.D.; Werner K. Gottstein, M.D., and Vincent A. Costanzo Jr., M.D., Chicago 172

Paresthesia of the Pharynx Due to Cervical Adenitis. I. M. Schnee, M.D., Paterson, N. J. 177

Bacillus Proteus Abscess of Cerebellar Lobe of Otogenic Origin: Report of a Case with Recovery. William R. Chambers, M.D., and William Clark, M.D., South Bend, Ind. 179

Dermoid Cyst of the Midline of the Neck. Charles F. Grabske Jr., M.D., Philadelphia 183

Squamous Cell Carcinoma Secondary to Cholesteatoma. Edwin H. Coachman, M.D., Muskogee, Okla. 187

Obliterative Frontal Sinusitis: Report of a Case and Review of Literature. Benjamin M. Volk, M.D., and S. Mukerji, M.B.B.S. (Patna, India), D.O.M.S. (London), Albany, N. Y. 188

CLINICAL NOTES; NEW INSTRUMENTS AND TECHNIQUES:

Nasal Packing: The Rubber Pneumatic Pack. Ralph W. Stevens, M.D., Walla Walla, Wash. 191

New Technique in the Use of a Surgical Tray. Joseph Jerome Littell, Indianapolis 195

Traction Sutures in Tonsillectomy. Rafael C. Tionloc, M.D., Manila, Philippines 198

Otosclerosis and ACTH: Preliminary Report. Zhivko Angeluscheff, M.D., New York 200

PROGRESS IN OTOLARYNGOLOGY. Summaries of the Bibliographic Material Available in the Field of Otolaryngology:

Functional Examination of Hearing. Alfred Lewy, M.D.; Sherman L. Shapiro, M.D., and Norman Leshin, M.D., Chicago 201

ABSTRACTS FROM CURRENT LITERATURE 223

DIRECTORY OF OTOLARYNGOLOGICAL SOCIETIES 230

THE A. M. A. ARCHIVES OF OTOLARYNGOLOGY is published by the American Medical Association to stimulate research in the field of otology and laryngology and to disseminate knowledge in this department of medicine.

Communications regarding subscriptions, reprints, etc., should be addressed, A. M. A. ARCHIVES OF OTOLARYNGOLOGY, American Medical Association, 535 North Dearborn Street, Chicago 10.

Manuscripts for publication, books for review and correspondence relating to contributions should be sent to Dr. George M. Coates, Chief Editor, 1721 Pine Street, Philadelphia 3, or to any other member of the Editorial Board.

Articles are accepted for publication on condition that they are contributed solely to the A. M. A. ARCHIVES OF OTOLARYNGOLOGY. Manuscripts must be typewritten, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones will be supplied by the Association when the original illustrations warrant reproduction and when their number is not considered excessive.

Footnotes and bibliographies (the latter are used only in exhaustive reviews of the literature) should conform to the style of the *Quarterly Cumulative Index Medicus*. This requires, in the order given: name of author, title of article and name of periodical, with volume, page, month—day of month if the journal appears weekly—and year.

Matter appearing in the A. M. A. ARCHIVES OF OTOLARYNGOLOGY is covered by copyright, but as a rule no objection will be made to its reproduction in a reputable medical journal if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the A. M. A. ARCHIVES OF OTOLARYNGOLOGY or in any of the other publications issued by the Association will not be permitted.

The A. M. A. ARCHIVES OF OTOLARYNGOLOGY is issued monthly. The annual subscription price (for two volumes) is as follows: domestic, $12.00; Canadian, $12.40; foreign, $13.00, including postage. Single copies are $1.25, postpaid.

Checks, money orders and drafts should be made payable to the American Medical Association.

OTHER PERIODICAL PUBLICATIONS
of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (three volumes): domestic, $15.00; Canadian, $16.50; foreign, $19.00. Single copies, 45 cents.

A. M. A. ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced original clinical and laboratory investigations in internal medicine. Illustrated. Annual subscription price (two volumes): domestic, $10.00; Canadian, $10.40; foreign, $11.00. Single copies, $1.00.

A. M. A. ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $12.00; Canadian, $12.40; foreign, $13.50. Single copies, $1.25.

A. M. A. ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY—Monthly. Devoted to advancing the knowledge of and progress in cutaneous diseases and syphilis. Publishes original contributions on these two subjects, transactions of the important dermatological societies, book reviews, etc. Illustrated. Annual subscription price (two volumes): domestic, $12.00; Canadian, $12.40; foreign, $13.50. Single copies, $1.25.

A. M. A. AMERICAN JOURNAL OF DISEASES OF CHILDREN—Monthly. Presents pediatrics as a medical science and as a social problem. Includes carefully prepared reviews, based on recent pediatric literature, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $12.00; Canadian, $12.40; foreign, $13.50. Single copies, $1.25.

A. M. A. ARCHIVES OF SURGERY—Monthly. Devoted largely to the investigative and clinical phases of surgery, with monthly reviews on orthopedic and urologic surgery. Well illustrated. Annual subscription price (two volumes): domestic, $14.00; Canadian, $14.40; foreign, $15.50. Single copies, $1.25, except special numbers.

A. M. A. ARCHIVES OF OPHTHALMOLOGY—Monthly. Includes original articles on diseases of the eye, annual reviews of special subjects, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $12.00; Canadian, $12.40; foreign, $13.00. Single copies, $1.25.

A. M. A. ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original articles and general reviews in the field of pathology. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, $1.00, except special issues.

A. M. A. ARCHIVES OF INDUSTRIAL HYGIENE AND OCCUPATIONAL MEDICINE—Monthly. Devoted to the advancement of knowledge of the diseases of industry and to the publication of scientific investigation in this field. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00, including postage. Single copies, $1.00.

QUARTERLY CUMULATIVE INDEX MEDICUS—A complete subject and author index to the worth while current medical literature of the world. Issued twice a year. Volumes bound for permanent reference. Subscription price, calendar year: domestic, $20.00; Canadian, $22.00; foreign, $22.00.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street **CHICAGO 10**

A. M. A.
ARCHIVES OF OTOLARYNGOLOGY

VOLUME 54 AUGUST 1951 NUMBER 2

COPYRIGHT, 1951, BY THE AMERICAN MEDICAL ASSOCIATION

MALFORMATIONS OF THE AURICLE AND THE EXTERNAL AUDITORY MEATUS

A Critical Review

FRANZ ALTMANN, M.D.
NEW YORK

AURICLE

IN VIEW of the development of the auricle, it is easily understood that one may find a great variability in the external configuration, particularly of the complicated relief of the auricle. These variations, however, are generally minute and may frequently be considered as physiological rather than pathological variations.

Anomalies in the Position of the Auricle.—With the head in an erect position, the external opening of the external auditory meatus is situated at the level of the upper margin of the ala nasi (Langer). Some old authors, and recently Desfosses, have concluded from the representation in works of art such as statues and paintings that in the ancient Egyptians the auricle had a particularly high location on the head. This assumption, however, has already been refuted by Langer. Examination of the heads of mummies shows that the auricle in ancient Egyptians had exactly the same location as it has today in members of the white race.

Little attention has been paid to the actual size of the angle of the external ear to the median plane. According to Linder, it averages 25 to 26 degrees in boys and 17 to 18 degrees in girls, 8 to 16 yr. of age. In both sexes the right ear often forms a somewhat greater angle to the median plane of the skull than the left ear (about 1 degree); in rare instances, this difference can reach comparatively high figures. In 362 out of 591 persons, however, the angles of the right and the left ear are equal. The upper part of the ear may protrude more than the lower part (Quelprud, 1932).

Frequently the auricle stands away from the head at an angle greater than normal. This condition, which may be combined with abnormal size of the auricle (macrotia), is called lop or bat or flaring ears. According to Young, the auricle can be divided into two parts—a medial portion, the concha, which is attached to the head, and a lateral, free portion, the scapha. The dividing line between the two portions is the anthelix. The angle between head and concha varies very little. It is the angle between concha and scapha which is considerably increased in lop ears while the relationship of the concha to the head remains unchanged. The anthelix is, as a rule, less prominent than in normal ears. A connection between the position

This work was done under grants from the Hayden-Coakley Fund and the Research Council of The American Otological Society.

From the Department of Otolaryngology, Columbia University, College of Physicians and Surgeons (Director E. P. Fowler Jr., M.D.).

and the relief of the auricle is also assumed by Geyer, who found that in primates as well as in man ears which stand out from the head at a greater angle have a less marked relief than those which are closely attached to the head. In certain races it is more usual for the ears to stand out from the head at a greater angle. This points to a constitutional origin of the condition. The hereditary trait is most probably recessive (Leicher).

A rare anomaly is the so-called abnormal adherence of the upper part of the auricle to the head (Gruber, Wreden, Ruttin, Šercer). While the lower half of the auricle stands out normally from the head, the upper half is adherent to it and the retroauricular fold is absent. The skin of the postauricular region passes directly into the skin of the lateral surface of the upper half of the auricle. It appears as though the upper part of the auricle had been pushed upward into a pocket of skin (*Kryptotie*, Šercer). The relief of the auricle and its cartilaginous skeleton is normal.

In these cases, the detachment of the posterior part of the auricle from the head, which usually occurs during the fourth embryonic month, fails to take place in the upper half. Lesser degrees of the same anomaly with only the anterosuperior part of the helix contained in the skin pocket are more frequent (*Taschenohr* [*Kubo*]—Otsuka, Noguchi and Kotake, Yamanake, Hanada, Kubo, Yokogawa).

Variations in the Relief of the Auricle.—The relief of the auricle is one of the most variable features of the human body. Many of the deviations from the normal form can be regarded as being within the normal range of variability. They represent individual variations rather than true malformations, although in many instances it is very hard to say that it is the one rather than the other. Many variations bear a resemblance to conditions normally present during the later embryonic stages of the human auricle and are regarded as persistence of these conditions. They resemble, on the other hand, regular findings in the auricle of certain monkeys.

In one group of these variations, which are frequently of a hereditary nature (Gaus, Powell and Whitney, Quelprud, and others), one finds on the upper part of the helix ascendens, a little above the level of the inferior crus of the anthelix, a hillock, the so-called tubercle of Darwin. This tubercle has been proved by comparative anatomic investigations to be homologous to the tip of the mammalian ear. According to the investigations of Schwalbe (1897), six degrees of the development of this tubercle can be distinguished, starting with its complete absence, presenting a slight thickening of the margin of the helix in other instances, and ending with the ears which show a resemblance to the auricle of certain apes (macacus-shaped and cercopithecus-shaped auricles). Regmunt-Sobieszczanski distinguishes seven different degrees; Quelprud (1934), eight. An unrolled portion between the top of the ear and Darwin's tubercle is found to be a sex-limited recessive, more frequent in men than in boys (Geyer, 1928). Bent-over helix, Darwin's tubercle, and adherent ear lobes are more frequent in women. The satyr point, a kink in the upper margin of the helix, normally persists to the second or third embryonic month, Darwin's point to the ninth month (Haike). Not infrequently, the crus helicis extends abnormally far back and may in some instances fuse with the anthelix, thus completely separating the cymba from the cavitas conchae (Langer, Féré and

Séglas, Davis and Phillips). The posterior end of the prolonged crus helicis may in rare instances be subdivided still further into two branches.

In another form a fold extends from the anthelix through the cymba to the helix (Langer). This is called crus anthelicis tertium (Schwalbe) or Stahl's ear (Binder).

If the anthelix protrudes above a plane passing through the tragus and helix, one speaks of it as Wildermuth's ear (Binder).

When the enlarged portion of the anthelix connects with the helix, it forms the Mozart ear, present in Wolfgang Amadeus Mozart and his father.

In the so-called "cat's ear," the auricle is folded forward and downward in varying degrees from above and behind.

"Cup-shaped" ears are a malformation in which the auricle is small; instead of lying parallel to the side of the head, it grows forward over the meatus to form a cup. The hereditary transmission is due to a single dominant gene and was observed through five generations (Potter).

In a mother and daughter, the helix was found by Haike to be flat in the upper part and connected with the anthelix.

In other cases, an abnormal configuration or a defective formation of the concha auriculae is seen. A vertical cartilaginous ridge in the concha, the so-called crus cymbae, is found in 5 to 6% of the ears. It is an irregular dominant character with very variable expression (Quelprud, 1934, 1939). At the back of the ear a projecting cartilaginous lump is sometimes present. It is more frequent in men than in women, but its manner of inheritance is unknown (Quelprud).

Aside from these more typical variations many atypical forms also occur.

The lobule of the auricle is, on rare occasions, entirely absent; in other instances it is abnormally large. Very often one finds adherent lobules which are sometimes extended towards the cheeks. Sometimes a free lobule in the newborn develops later on into an adherent one (Geyer). Adherence or absence of ear lobes are a characteristic of Bushmen and Tierra del Fuegans; they are often absent in Negroes and Filipinos. The frequency of adherent ear lobes in Germany is 10 to 25% (Hildem). They are commoner in women than in men (Riddell).

The free lobule shows a dominant, the adherent lobule a monomeric, recessive trait (Hilden, Leicher, Geyer, Powell and Whitney, and others).

Variations in the finer relief of the lobule are very frequent. In rare instances, one finds various degrees of a bifid lobule ranging from a slight indentation to a complete division into two parts. It must be distinguished from a traumatic cleft caused by the wearing of heavy earrings. The congenital cleft is thought to be caused by the lack of developmental junction of the parts of the mandibular and hyoid arch or the hyoid arch alone which form the lobule (Aprile, Scalori, and others). According to Schofield, it shows an imperfectly dominant hereditary trait.

Hypertrichosis of the auricles has been described by Tommasi. The condition starts around puberty and eventually the entire outer and lower half of the inner surface of the auricle becomes covered with long black hair. The abnormality was confined to males through five generations. Its holandric inheritance follows the y-chromosome, which must therefore contain the determining gene (Gates).

True Malformations of the Auricle.—True malformations, particularly the severe forms, are often found in combination with malformations of other parts of the ear or with malformations of the face.

In rare instances, the auricle is divided into two parts by a fissure (Coloboma auriculae). The fissure runs either vertically and divides the auricle into an anterior and a posterior part (Marx; less severe cases—Eggston and Wolff) or it runs in a transverse direction and divides it into a superior and an inferior part (Gruber, Keith). These malformations are explained by anomalies in the closure of the first external branchial cleft and in the union of the parts of the auricle which are derivatives of the first branchial arch with those of the second branchial arch. The congenital "slit-shaped" auricular fistulas described in two cases by Onodi are possibly the mildest degree of a transverse fissure. In these cases, a slit was located below the crus helicis and extended 5 to 7 mm. from there into the anterior wall of the external auditory meatus.

Abnormally large size of the auricle is called macrotia. Undersize of the auricle is called microtia. Macrotia is usually bilateral, but in rare instances unilateral (Moraza, Podgaetsky). There are considerable racial differences in the size of the auricle. According to Bean, Negroes have the smallest and members of the Asiatic yellow-brown races the largest ears. The ears of the white race are intermediate, being longer than those of the Negro race but just as broad. Macrotia (lop ear) is frequently formed as a hereditary character in domestic animals. Its development in rabbits has recently been analyzed by Castle and Reed and by Dunlop and Hammond. More marked degrees of microtia are combined as a rule with malformations of the auricle. Marx divides these cases of microtia into three groups, according to the degree of the malformation. In the first degree, the different parts can be recognized; a real defect is not found. In the second degree, the rudiment of the auricle is represented by an oblong ridge which resembles the helix in configuration and condition. In the third degree, different parts cannot be differentiated in the rudiment; it is represented by an irregular uneven ridge or by one or more hills. In very rare cases, there is a complete absence of the auricle (anotia). Seven such cases have been reported so far (Colomiatti, Chiari, Iwata, Krampitz, Lynch, Šercer, Ružic).

There is a close connection between the development of the auricle and the mandible, there being a parallelism between the formation of the mandible and the gradual moving of the auricle craniodorsally from its original caudoventral position. When one considers the frequent combination of malformations of the ear and the face, especially of the jaw, one can easily understand that the malformed auricle frequently has a more caudoventral position than normally. This condition is called melotia.

Auricular malformations also occur in animals and there is in many instances definite evidence of heredity. In most cases there was no anatomical examination of the malformed areas, and very often no mention is made whether the auricle alone or also the external canal and the deeper parts were affected. For that reason the two groups must be reviewed together.

In certain sheep races individuals with short auricles or even without auricles but with normal external auditory meatus are not uncommon. The anomalies follow a dominant hereditary trait, the character absent ear being homozygous, the character short ear being heterozygous (Ritzmann, Wriedt, Wassin, Lush).

Similar conditions seem to prevail in certain goat races (Wassin).

Agenesia of the ear combined with other malformations, such as cleft palate, shortening, and ankylosis of the mandible, in Norwegian sheep, was described by Mohr (1929). It is a recessive and lethal anomaly.

Anomalies of the auricle alone in cattle with a dominant hereditary trait were described by Yamane, Lush, and Snyder. Absence of the auricle is mentioned by Weber.

Short ears in rabbits with normal external auditory meatus combined with brachydactylia following a simple recessive hereditary trait were described by Greene and Saxton. Hopf mentions a litter of rabbits which contained one with a single ear. From it a strain of one-eared rabbits was produced.

Absence of the auricles in hares is mentioned by Gradenigo and Lönnberg; microtia in horses, by Zimmermann.

In pigs, small size or absence of the auricles and bilobed auricles were found as single malformations or together with atresia of the external auditory meatus and other ear defects, with defects of other parts of the skull, cleft palate, and other anomalies. The genetics of these malformations have not been clarified yet (Meyer, Zimmermann, Nordby, Idaho Agricultural Station, Annett, Buchanan, Robinson and Bryant, and others).

Extensive studies on the heredity of ear malformations were carried out on mice, which invariably showed a recessive hereditary trait.

The following anomalies are known: 1. Short ears (Lynch) with a marked retardation of pinna growth, whereby the ear of an adult mouse looks like that of a 2-wk.-old mouse. The ear malformation is combined with skeletal abnormalities (Grüneberg). 2. "Hounds ear" (McPheters and Little). 3. "Lowering of the auricle with inversion of the pinna and crop ear" (Kobozieff and Pomriaskinsky-Kobozieff). 4. A character with very variable expression described by Feldman:

> The least visible effects are a slightly smaller pinna than normal. The pinna may show any degree of inhibited development to the point where it is only a small band of tissue. Such extreme inhibitions of development of the pinna are accompanied by defective middle and inner ears. In very defective mice other parts of the head, especially the eye, are affected.

Auricular Appendages.—A frequent group of malformations are the so-called auricular appendages or accessory auricles. They are found in about 1.5% of the population and are more frequently unilateral than bilateral. To some extent they are undoubtedly subject to heredity. Although no definite opinion can be formed as yet as to the exact method of transmission, many circumstances speak in favor of a dominant predisposition with variegated penetrance (Brander).

The auricular appendages usually have the shape of hills or flaps, sometimes having pedicles. They vary from the size of less than half of a pea to that of a hazelnut; they may be single or multiple. The appendages are usually situated just in front of the tragus or ascending part of the helix, often in direct connection with these structures. In a case reported by Jenkins a "skin papillus" with a diameter of about 4 cm. was seen in front of the left tragus. It was found six times in four generations of the same family and was transmitted as an irregular dominant by two men who did not show the condition.

According to Jones and Wen, the line of predilection of the appendages lies just in front of the line of distribution of the preauricular fistulas, which will be discussed later. This line extends from the temple to the anterior margin of the

crus helicis and the interspace between the latter and the upper part of the tragus, thence into the concha to the external auditory meatus, and out again between the lower part of the tragus and the antitragus to the junction between concha and cheek. This line corresponds, according to the authors, to the line of junction between the hyoid and mandibular folds. The auricular appendages result from excessive growth in the mandibular fold in the neighborhood of this line. As a rule, the appendages are found in this location in ears which are otherwise normal.

Less frequently, they are also found farther away from the auricle in a zone extending from the lower end of the just described line to the angle of the mouth. This zone corresponds to the line in which the maxillary and mandibular processes of the first branchial arch grow together. Appendages in this location are more frequently combined with other malformations, such as microtia, melotia, or transverse fissure of the cheek. They are evidently due to disturbances in the union of the two processes of the first branchial arch.

As Marx points out, one is able to infer a morphologic sequence from the severest malformations to almost normal conditions: persistent transverse fissure of the cheek with auricular appendages, transverse fissure extending over the entire cheek but irregularly cicatrized during intrauterine life with auricular appendages, irregularly cicatrized remnants of the dorsal part of the fissure with appendages, and isolated appendages.

In very rare instances, the appendages are numerous and may be grouped so that they form a structure which resembles to some extent a normal auricle, in which case we speak of polyotia. Although several cases of this kind have been reported, only the case of Bol and de Kleyn can be regarded as true duplication of the auricle (Kahler). In a newborn baby there were on one side two well-built auricles facing each other like reflected images and grown together along a line extending from the incisura anterior to the incisura intertragica. The tragus was absent. On the other side, there were two preauricular appendages, but the auricle was normal.

The appendages commonly contain variously shaped but usually oblong pieces of elastic cartilage. Of special practical interest is the fact that these cartilaginous parts frequently extend deep into the underlying tissues. This must be kept in mind when one is attempting surgical removal, for otherwise one might be unpleasantly surprised and might even find it difficult to avoid injuring branches of the facial nerve.

Congenital Auricular Fistulas.—Of great practical importance and not excessively rare are congenital fistulas (sinuses, fossa pits) of the ear. Selkirk found them in 0.9% of 3,600 white and in 5.2% of 346 Negro children. In 22.7% of the children the fistulas were bilateral. A preauricular pit was found in 10 to 14% of Asiatics by Ride. They are represented by blindly ending narrow tubes, sometimes by mere pits or dimples. They are most frequently found along the above-described line of predilection of Jones and Wen.

Congdon, Rowhanavongse, and Varamisara divide the fistulas into several groups according to their location. By far the most frequent location (about 90% of Congdon's cases) is the marginal helicine location, where the outer opening of the fistula is close to the anterior border of the ascending limb of the helix. Next in frequency are the preauricular fistulas. Most of them lie in a line extending

from the lower border of the crus helicis toward the angle of the mouth, and many of them close to the marginal helicine field. The majority of these fistulas are associated with congenital scars. In other cases such scars have been found even without fistulas. Not uncommonly, a small pigmented mole has been seen in front of and a few centimeters from the fistulous opening (Aird). Other locations of fistulas are crural, in the crus helicis; posterior helicine, in the middle part of the helix descendens; helicolobular, at the border between helix and lobule; postauricular, with the opening of the sinus behind the auricle and close to its attachment at the level of the tragus. Many of the reported postauricular fistulas, however, are apparently secondary to infection rather than due to faulty development (Selkirk). The last group comprises the central lobular fistulas, situated in the middle of the lateral surface of the lobule, associated with a similar pit at a corresponding portion on the medial surface of the lobule. A communication between the two openings is rare (Sutton, Jenks). The appearance of these "natural pits," or "earring holes," may be indistinguishable from that of old healed earring puncture marks. Sometimes pits appear only on the medial side (Scalori) or more rarely only on the lateral side of the lobule (Edmonds and Keeler). Edmonds and Keeler describe two pedigrees of the condition. In one of them there was dominant heredity; in the other, either recessive or dominant with low penetrance.

Aside from these typical sites, fistular openings are found in the fossa triangularis, in the cavitas conchae at the level of the tragus, and in the antitragus. The fistulas vary in length, are quite often very deep, may take a tortuous course, and show ramifications. In the case reported by Zöllner, a typical marginal helicine fistula extended upward and back and eventually down behind the ear, terminating over the mastoid.

In rare instances, fistulas have been observed extending from the floor of the external auditory meatus, laterad from the posterior belly of the digastric muscle, downward to the angle of the mandible. The upper opening was in either the deep or the superficial parts of the meatus, sometimes between tragus and antitragus. The lower opening was behind the angle of the mandible, near the anterior border of the upper half of the sternocleidomastoid muscle.

Of these "colloaural" fistulas, the following have reported cases: Harding, König, Hirschfeld, Flint, Küttner, Fournier, Cauchoix, Bergara and Bergara, Seebohm, Oppikofer.

In two other cases (Caldera, Wildenberg), the lower opening was not present at birth but developed secondarily after abscess-formation in the lower part of the duct.

Riedel and de Ruyter, and recently Leisse, report cases of "colloaural" fistulas in which the upper end of the fistulous tract opened into the middle ear. This is questioned, however, by Oppikofer, who thinks that it opened rather into the fundus of the external auditory meatus.

It seems doubtful that Oppikofer's objections can be justified, because fistulas might actually open into the middle ear. This was proved in histological serial sections by Druss and Allen. They report a case of "colloaural" fistulas with bilateral microtia in which preauricular fistulas went backward and downward and opened on the neck immediately behind the lower margin of the auricle. The lower

end of this fistulous tract marked the beginning of a second tract, which extended into the depth and opened into the middle ear.

In the frequently mentioned case of Virchow, there was a right-sided microtia with melotia and rudimentary external auditory meatus. Immediately below the auricle there was an auricular appendage and below that a fistulous opening through which a probe easily penetrated into the posterior tonsillar pillar just behind the upper pole of the tonsil.

Auricular fistulas are, in the majority of cases, isolated malformations. Sometimes, however, they are combined with other malformations, such as auricular appendages (Schüller, Seifert, and others), microtia, branchiogenic fistulas of the neck (first described by Heusinger and reported also by Carp and Stout, Hyndman and Light, Guyot, Roques-Sativo, Baumgartner and Steindel, and others), fistula in the anterior tonsillar pillar (Levinstein), thyroglossal cysts (Selkirk), fistulas of the naso-optic fissure (Reh), median nasal fistula (Nager), microphthalmia, and other malformations of the bulb (Franceschetti and Valerio), harelip, and cleft palate, albinism in Negroes (Pipkin).

A typical combination of preauricular fistulas and other ear deformities with malformations of facial bones and other structures derived from the first visceral arch was first described by Franceschetti and Zwahlen in 1944 under the name of "mandibulofacial dysostosis." It consists of the following features (Franceschetti and Klein):

1. Palpebral fissures sloping downward laterally ("antimongoloid") with a coloboma in the outer portion of the lower lids and, more rarely, in the upper lids.
2. Hypoplasia of the facial bones, especially the malar bones and mandible.
3. Malformations of the external ear and occasionally the middle and inner ears. The auricle usually occupies a low position.
4. Macrostomia, high palate, abnormal position, and malocclusion of the teeth.
5. Blind fistulas between the angles of the mouth and the ears.
6. Atypical hair growth, in the form of tongue-shaped processes at the hairline, extending toward the cheeks.
7. In some cases other associated anomalies, such as facial clefts and skeletal deformities.

According to the severity and symmetrical extension of the syndrome, it can be subdivided into completely developed, incomplete, abortive, atypical, and unilateral forms.

The condition is due to an inhibitory process occurring at the end of the sixth or the beginning of the seventh week of embryonic life affecting facial bones and other derivatives of the first branchial arch.

The syndrome is genic in origin and transmitted in an irregular dominant mode. One is dealing with an unstable gene of occasionally pleotropic (polyphaenic) effect, especially on the skeletal system. There seems to be a disturbance of organizer action, probably due to a lethal or sublethal effect of the gene concerned (Franceschetti and Klein).

A number of cases had been described before they had been recognized as representative of a special syndrome (Berry, Collins, Pires de Lima and Monteiro, Isakowitz, Waardenburg, Hermans, van Lint and Hennebert, McEnery and Brennemann, Debusmann, Kilner, Johnstone, Mann). After the publication of the paper of Franceschetti and Zwahlen, cases were reported by the following authors:

Leopold, Mahoney and Price, Schachter, Bregeat and Naud, Brohm and Kluska, Holm, Navis, Sanvenero-Roselli, Franceschetti and Klein, Waardenburg and Navis, Straith and Lewis, Gayral, Bru and Paintandre.

The auricular fistular ducts are lined with stratified squamous epithelium and are provided with a wall of connective tissue in which hairs, sebaceous glands, sudoriferous glands, and, in rare instances, an island of cartilage (Aird) may be found embedded. If the fistulas are dry, they cause no complaints; however, they often excrete a small amount of white fluid which may cause eczematous changes of the surrounding skin. Sometimes the mouth or other parts of the fistula may close, with retention of the content and often with subsequent formation of an abscess. In such cases, when surgical intervention is necessitated, general surgical principles must be applied. When treating fistulas which are not acutely inflamed, one must remember that often they extend rather deeply. Because of this, obliteration of the fistulas with chemical sclerotics or electrocoagulation frequently does not result in permanent cures. When surgical removal of the fistulous tract is attempted, determination of its extent by injection of methylene blue or other coloring substances (Baumgartner, Dingman, Miller and Moore) seems advisable. Owing to the branching of some of these fistulous tracts, their surgical removal by sharp dissection is often extremely difficult and sometimes impossible. Surgical diathermy is probably the most effective method of treating complex preauricular fistulas (Pastore and Erich) after exposure of the fistulous tract and all its ramifications with the electric scalpel (Havens).

Frequently an increased occurrence of congenital fistulas in certain families has been reported. It is possible, at times, to trace fistulas through several generations of the same family (Schüller, Montgomery, Selkirk, Powell and Whitney, Connon, Fox, and others). Sometimes auricular appendages are seen in one member of a family and auricular fistulas in other members (Ruttin). It is more or less generally agreed that the hereditary trait is produced under the influence of an incompletely dominant factor of variable manifestations and without sex difference (Kindred, Selkirk, Potter, Whitney, Edmonds and Keeler, Mengele, Quelprud, Connon, McDonough, Stiles, and others). In the unilaterally affected members of any given family, one side, the right or left, is more frequently affected than the other side (Edmonds and Keeler). Preauricular fistulas are found in numerous domestic animals, such as horse and sheep, even more frequently than in man (Moldenhauer, Sington, Keith).

The genesis of the fistulas is evidently not uniform. Those situated along the line of junction between the mandibular and hyoid arch ("line of predilection") could very well be derived from remnants of the first external branchial cleft as emphasized by Jones and Wen. Those extending forward and downward toward the angle of the mouth seem to be caused by disturbances in the union of the mandibular with the maxillary process of the first branchial arch. The close relationship between these fistulas and the auricular appendages seems easily understandable.

The "colloaural" ("hyomandibular") fistula is also caused by irregularities in the closure of the first external cleft (Küttner, Seebohm, Oppikofer). According to Küttner, the fistula takes its origin from remnants of the first, or hyomandibular, cleft, and its course coincides with the upward and backward migration of the

auricle during the embryonic development. In 20-cm.-embryos, the auricle is situated at a place which in later life would correspond with the angle of the mandible.

The fistulas and pits in other parts of the auricle, such as posterior helicine, postauricular, and lobular fistulas, are most probably caused by a defective junction of certain auricular hillocks. Although it must be conceded that some of the fistulas within the "predilection line" could have the same origin, this cannot definitely be true of all the fistulas in these locations, as assumed by some authors (e. g., Congdon and co-workers, Guyot).

The fistula described by Virchow was combined with microtia and a rudimentary external auditory meatus and opened into the pharynx. According to the majority of authors, it is derived from the first branchial cleft. The thin epithelial membrane which at one stage of development separates the first external from the internal cleft is supposed to have been torn in this case. According to Wenglowski and Oppikofer, however, Virchow's case represents an ordinary complete lateral fistula of the neck combined with melotia and congenital atresia. In cases in which a branchiogenic fistula of the neck communicated with the middle ear (Druss and Allen, and possibly Riedel, Ruyter, Leisse) a more complicated branchiogenic origin must be assumed.

Congenital Arteriovenous Fistula.—Congenital arteriovenous fistulas, or cirsoid aneurysms, are sometimes found in the auricle, the external auditory canal, or the middle ear (Smith, Lenzi, Maggiolotti, Gutierrez, Montserrat, Horton and Hempstead, Kepes, Faleeva, Luke, Chimani, Pirodda, and others). They consist of multiple arteriovenous anastomoses resulting from an anomaly of the vascular system. The anastomoses are formed either between internal carotid artery and jugular vein or between smaller arteries and veins in the auricular region. They possibly develop from congenitally malformed arteriovenous anastomoses (Clara). The regular presence of such anastomoses in the auricle has, as already mentioned, been made probable by the fact that typical glomus tumors have been reported in this region (Sannicandro, Ertl).

Congenital Tumors.—In addition to the malformations cited here, there are several congenital tumors of the auricle which occur in rare instances. They are lymphangiomas, hemangiomas, papillomas, nevi, epidermoids (Citelli, Hager), and true dermoids. The true dermoids are generally located behind, or behind and above, the insertion of the auricle. They are usually connected with the periosteum. In rare cases they are situated on the auricle itself, even on the lobule or in the preauricular region (Fano, Breul, Goldmann, Halász, Johnston, Levesque, Joseph, Jacques and Aubriot, German, Cini, Gritti, Comes, Heck, Edmonds and Keeler, and others).

EXTERNAL AUDITORY MEATUS

The external auditory meatus consists at birth of an outer cartilaginous part derived from the primary meatus and an inner membranous part derived from the meatal plate. It obtains its definite form during the first years of postnatal life mainly through the development of the osseous canal, which replaces the inner membranous part. The anterior and inferior walls as well as the inferior part of the posterior wall are derived from the os tympanicum, while the main parts of the posterior and superior wall are built from the arcus zygomaticus and the mastoid

process. At birth the tympanic bone is represented by an imperfect ring which is open above. The ossification of the anterior and inferior walls does not take place equally by continuous apposition of bone, for, as a rule, one finds more intensive formation of bone at two points, one posteriorly and one anteriorly. These points of more intensive growth join in the second year and border a bony defect, which is usually closed by the fifth year but may persist into later life. This is the case in 19% of the skulls of adult Europeans; in certain races the ossification defects are much more frequent. In South American Indians, e. g., they are found in 54% of the skulls (Möller-Holst).

Changes in the Shape of the Meatus: Individual and Anthropologic Variations.—There are remarkable individual and also racial differences in the formation, especially of the width, of the external meatus. In some races the meatus is surprisingly often found to be considerably narrowed. The narrowness is due either to a more or less diffuse hyperostosis of the upper free ends of the tympanic bone, particularly the posterior end, or to more circumscribed exostoses in the innermost part of the osseous canal. These exostoses are located near the annulus tympanicus, at the anterior or posterior margin of Shrapnell's membrane, at the floor, or nearer the anterior or posterior wall of the meatus. Sometimes, in addition, exostoses which originate from the external wall of the squama of the temporal bone are found just above Shrapnell's membrane. In many instances these two types—hyperostoses of the outer parts and exostoses of the innermost part of the osseous canal—are combined. In view of the numerous transitory forms, a clear distinction between exostoses and hyperostoses is not always possible.

The condition develops chiefly during later adolescence and the earlier half of adult life; it is most frequently bilateral and very often symmetrical. When it is one-sided, it occurs more commonly in the left than in the right ear. Males are considerably more subject to the growths than females. The exostoses of the innermost part of the meatus are believed to originate from abnormalities in the ossification of the annulus tympanicus which do not become manifest until later in life. It has been assumed that at certain points a predisposition to the formation of new bone prevails which eventually leads to the formation of the exostoses (Ruttin).

The exostoses have for a long time aroused the interest of the anthropologists as well as of the otologists. This can be seen by the great number of publications on this subject (Seligmann, 1864, Welcker, Wyman, Virchow, Turner, Blake, Ostmann, Ferreri, Burton, and many others). In recent years several comprehensive papers were published by anthropologists (Möller-Holst, Hrdlička) as well as by otologists (Alexander, Ruttin).

A "predisposition" to ear exostoses is now probably universal in man, but in some races or groups the formation of abnormalities, owing perhaps to direct hereditary effects, is much more frequent than in others. The highest frequency of the condition is found among the old aborigines of the North American continent and in Polynesia. They are also frequent in some parts of Mexico and South America (Peru, Bolivia). Europeans, who are much less frequently affected, have, on some occasions, shown a familial occurrence of these exostoses and hyperostoses with apparently a dominant hereditary trait (Kessel).

In populations with a predisposition to their formation exostoses occur in less than 20 per cent of the people. They should therefore not be regarded as racial

characteristics (*Rassenmerkmale*) but only as racial peculiarities (*Rasseneigentümlichkeiten*, Möller-Holst).

The etiology of these exostoses and hyperostoses has been widely discussed but not clarified as yet. According to Hrdlička, one must distinguish between systemic or predisposing and exciting causes of the condition. The exciting cause, when the predisposition exists, may be anything mechanical or chemical that produces prolonged irritation with consequent hyperemia or inflammation of any part of the bony meatus. Climate can be ruled out as an etiological factor because it has been found that, although the frequency of the condition varies very considerably in different territories, there is no correlation between exostoses and climate or other geographical factors.

A number of authors (among them recently van Gilse, Belgraver, Hlavaček, Meyer, H., and Adams) regard irritation by water, particularly by salt water, as a cause of ear exostoses in some instances. Some efforts were made to connect the frequency of ear exostoses in the Peruvians and the Channel Island Californians with sea bathing. It was not known that the abnormalities would be found to be even much more frequent in some of the inland tribes; that they occurred also in parts of Peru where no one bathed; that they were rare in ancient Egyptians who were great water lovers, and that the Malays and the boys of other marine groups, including certain whites, were great swimmers and divers without being particularly troubled with ear exostoses.

Notwithstanding the above, sea water is undoubtedly capable of causing chronic ear troubles, and these may conceivably act, where there is a tendency to exostoses, as exciting or favouring causes. (Hrdlička.)

The findings of Fowler and Osmun in guinea pigs with new formation of bone on the inner surface of the bulla tympanica after irrigation of the external canal with cold water could possibly be explained in the same way. According to O. Mayer, the hyperostoses develop from mechanical irritation of the tympanic bone and of the tympanosquamous fissure during swimming. The irritation is caused by the overstretching of the head and the pushing forward of the lower jaw in order to keep the mouth and nostrils as high as possible.

For understanding the possible causes of the predisposition, the following facts must be kept in mind, according to Hrdlička.

From all indications, exostoses are found only in man and not in any anthropoid apes, any other primates, or mammals. So far they have not been found in early (geologically ancient) man. The oldest known skulls with exostoses are from the neolithic period (Miyake). The frequency of the condition in the prehistoric American skulls, however, is a certain reminder that the beginnings of the affection are much older. Thus, one can say that the exostoses are confined to recent and present man.

The development of every part of the body is under a very definite and heredity-bound neurovascular control. In apes and lower forms such control of the external auditory meatus is evidently thoroughly established and fully adequate. In man, this control appears to be disturbed and no longer wholly sufficient; and with or even without sufficient exciting causes abnormal bony growths in the ears are the result. Such a disturbance or weakening could possibly be an accompaniment of the unexpectedly great and rapid evolution of the human head. Such radical change

must have disturbed preexisting trophic controls, and a full reaccommodation has not been reached, at least not in most of the human groups.

Within some 500,000 years man's progress has far outstripped, especially as concerns his brain and head, that of all the rest of creation. The rapid progress and differentiation with a spread to all regions and exposure to a multitude of new factors, has prevented in many respects a full adjustment of all parts, a full harmonization and stability in all regions. There is a possibility that the central trophic control of the external meatal region in the greatly enlarged, altered, and still altering skull, has not regained the full life-long adequacy that it possessed before. This would mark the abnormality under consideration as an incidental condition, one that might disappear in the natural course of events, if further skull changes affecting the part stopped and if direct inheritance of the anomaly did not meanwhile become rooted. Should this conception prove to be true, then the process of ear exostoses could be defined as that of irregular outgrowths of bone in the external auditory canal and principally from its tympanic part, due primarily to evolutionary weakened normal neuro-vascular control of the parts. (Hrdlička.)

In addition to the types of exostoses just discussed, the so-called solitary exostoses must be mentioned. They originate with a pedicle of varying thickness from the posterosuperior and, in exceptional cases, from the anterosuperior wall of the outermost part of the osseous meatus. They are always unilateral, not combined with any other exostoses or hyperostoses of the meatus, and covered with a thick grayish skin, and they attain considerable size; often they are visible in the external opening of the meatus. In this group one never finds any evidence of heredity. Macroscopically as well as histologically they resemble the osteomas of the paranasal sinuses, and both are possibly derived from dislocated periosteal "germs" or "rests" (Ruttin).

Most of the anthropologists do not recognize a distinction between the exostoses and hyperostoses and the solitary osteomas. They feel that both groups represent the same condition and that the differences are only gradual but not essential (Möller-Holst, Hrdlička).

Clinical experience, however, definitely favors such a distinction and my own experience tends to confirm the views expressed by Ruttin and other clinicians.

The most interesting aspect of the problem of the ear exostoses is, in my opinion, the relationship which seems to exist between it and the problem of otosclerosis.

Both exostoses and otosclerosis are conditions confined to man. Otosclerosis is, according to Guild, eight times less frequent in Negroes than in members of the white race; African Negroes are, at the same time, nearly free from exostoses (Hrdlička). Mayer's claim that both conditions represent tumor-like malformations (hamartomas) which not infrequently occur simultaneously and might also be combined with malformations of the inner ear has not been substantiated. According to a later hypothesis of Mayer, the otosclerotic foci develop in those regions of the labyrinthine capsule which are particularly subjected to mechanical tensions. Normal temporal bones are able to bear this strain. Others, however, show a lessened resistance, most probably on the basis of a constitutional inferiority of the bone. In these cases, otosclerotic foci develop in the areas of the greatest tension in an attempt to reinforce these regions. They are comparable to Looser's areas of reconstruction (*Umbauzonen*) in the long bones. Otosclerotic foci, therefore, are manifestations of a discrepancy between the forces acting upon the capsule and those of static resistance (*statische Widerstandsfähigkeit*) of the capsule in constitutionally inferior temporal bones.

A complete analysis of the forces acting upon the human temporal bone is lacking; an analysis of the same forces in animals has hardly been started (Loebell and Nauck).

Unquestionably, great differences exist between man and the other animals because of two factors: the enlargement of the brain in man, together with the changing relationship between brain case and facial skeleton, and the change from the quadruped to the upright gait.

In analogy to Hrdlička's assumptions that the disturbances of the neurovascular control of the external auditory meatus may lead to exostoses, disturbances in the mechanical adjustment of the skull, particularly of the temporal bones, may lead to the formation of otosclerotic foci. This theory would explain why the foci are found only in man.

Just as with the exostoses, only the constitutional predisposition to their development, but not the foci themselves, are inherited. Both conditions can, in the last analysis, be traced back to disturbances in the adjustment of the skull to relatively rapid changes in the course of phylogenetic development. The two conditions may or may not be simultaneously present but are independent of each other.

Hrdlička's suggestion that the tendency toward the formation of exostoses might in the course of the further phylogenetic development eventually disappear after further skull changes have stopped and the adjustment to them has become complete, unless direct inheritance of the condition did not meanwhile become the rule, could also be applied to otosclerosis.

I am fully aware of the fact that the ideas just outlined are of a purely speculative nature. They may, however, be of some value in our search for a solution of one of the greatest puzzles in otology, the question of the cause of otosclerosis.

Mention should finally be made of the fact that in Eskimos the entire tympanic bone is eccentrically thickened; the lumen of the osseous canal, however, shows normal width. This thickening is a regular finding and does not represent a pathological hyperostosis but a racial characteristic of the Eskimo skull (Möller-Holst, Stewart).

CHANGES IN THE SHAPE OF THE MEATUS DUE TO TRUE MALFORMATIONS

Changes in the shape of the external auditory meatus exceeding the physiologic or anthropologic variability are rarely found as isolated malformations. Because of the different origin of the two parts of the external meatus, one will find that deformities do not always affect both parts, at least not in the same degree; for instance, excessive narrowing of the cartilaginous meatus, which is supposed to be a hypoplasia of the primary meatus, has been observed. Sometimes the physiologic narrowness between cartilaginous and osseous meatus (between primary meatus and meatal plate) is more marked than normally. In some cases, one finds a hypoplasia of the entire anlage.

In rare instances, the lumen of the external canal is divided by a septum into two parts. Although in the great majority of cases the septum is a residuum of preceding inflammatory changes, a few observations have been reported in which its congenital nature is very probable. The majority of them showed, in addition, auricular appendages. In one group of cases (Brieger, Guranowski, Habermann) the lumen of the cartilaginous meatus was divided into two parts; one showed a

normal course and terminated at the drum membrane; the other was much shorter and ended blindly. These changes are more easily explained by anomalies of the first anlage of the primary meatus than by anomalies of the closure of the first branchial cleft, as assumed by Marx, Selkirk, and others. In Spira's case, in which the duplication of the outer part was combined with a lack of the osseous canal, an additional lack of the anlage of the meatal plate must be postulated.

In an observation of Walde, a ridgelike septum was seen on the floor of the osseous canal which at the drum extended up to the umbo and, in a case reported by Oppikofer, up to the upper margin of the drum. Anomalies in the anlage of the meatal plate explain this malformation.

The severer malformations are usually combined with malformations of the middle ear or of the auricle. We find either a defect of the inner part alone, caused by lack of the anlage of the meatal plate, or a defect of the inner part together with rudimentary or absent anlage of the primary meatus. Of special interest are those rare cases in which the inner part of the meatus is present but the external part is absent (Kaufmann, Case *2*; Alexander and Benesi, Case *2*; Altmann, Case *2*). As the growth of the meatal plate can take place only if the primary meatus has been developed, one must assume in these cases a secondary disappearance of the primary meatus. When one considers that the meatal plate is formed over a longer period by a solid epithelial mass, it seems possible, from an embryologic point of view, that in some places the central epithelial layers may fail to disappear and a secondary ingrowth of the mesenchyme may take place; in this way a stricture (Landauer) or a membranous closure of the meatus may develop at the border between the primary meatus and the meatal plate (Krampitz). Such congenital strictures and closures are, however, much less common and much less important than secondary inflammatory closures of the meatus caused, for example, by chemical trauma or by extension of a necrotizing acute otitis media into the tissues surrounding the external meatus.

Congenital Tumors.—In rare instances, dermoid cysts (Antognoli, Kaplan and Donlon) or teratoid tumors originate from the wall of the osseous canal. The latter contain various derivatives of all three germinative layers (Adam and Gilmour) or teeth (Becco, Marshall). Lüscher has reported a chondroma which originated from the anterior wall of the osseous meatus and consisted of elastic cartilage. He thinks that it is of congenital origin and regards it, analogous to the auricular appendages, as a derivative of the first branchial arch. A chondroadenoma has been described by Yokogawa.

BIBLIOGRAPHY

The following bibliography contains the available literature since 1930 and selected more important earlier publications. The papers denoted by an asterisk are not especially mentioned in the preceding article.

Adam, J., and Gilmour, M. D.: Teratoid Tumor of External Auditory Meatus, J. Laryng. & Otol. **45**:550, 1930.

Adams, W. S.: The Aetiology of Swimmers' Exostoses of the External Auditory Canals and of Associated Changes in Hearing, Proc. Roy. Soc. Med. **42**:424, 1949.

Aird, I.: Ear Pit (Congenital Aural and Pre-Auricular Fistula), Edinburgh M. J. **53**:498, 1946.

Albrecht, W.: Über Konstitutionsprobleme in der Pathogenese des Hals-, Nasen-, und Ohrenkrankheiten, Ztschr. Hals-, Nasen- u. Ohrenh. **29**:18, 1931.

*Der Einfluss der Konstitution auf die Erkrankungen von Hals, Nase und Ohr, ibid. **40**:309, 1937.

*Erbbiologie und Erbpathologie des Ohres und der oberen Luftwege, in Just, G.: Handbuch der Erbbiologie des Menschen Vol. 4, Berlin, Springer-Verlag, 1940, Pt. 1, p. 1.

Alexander, G.: Zur Kenntnis des Schlaefenbeines des amerikanischen Indianers, Montasschr. Ohrenh. **64**:237, 1930.

——and Benesi, O.: Zur Kenntnis der Entwicklung und Anatomie der kongenitalen Atresie des menschlichen Ohres, ibid. **55**:195, 1921.

Altmann, F.: Zur Anatomie und formalen Genese der Atresia auris congenita, ibid. **67**:765, 1933.

Annett, H. E.: A New Defect in Pigs: An Apparent Lethal Factor, J. Hered. **29**:469, 1938.

Antognoli, G. C.: Cisti dermoide del condotto uditivo esterno, Ann. laryng., otol. **35**:77 and 87, 1935.

Aprile, V.: Coloboma del lobulo dell'orecchio: Contributo alla studio del suo significato embriologico, Atti. Clin. oto-rino-laring. Univ. Roma **31**:293, 1933.

Baumgartner, C. J.: Bronchial and Thyroglossal Duct Cysts and Fistulas in Children, Surg., Gynec. & Obst. **56**:948, 1933.

——and Steindel, S.: Differential Diagnosis Between Thymic Duct Fistulas and Branchial Cleft Fistulas: Report of Case of Bilateral Aural Fistulas and Bilateral Thymic Duct Fistulas, Am. J. Surg. **59**:99, 1943.

Bean, R. B.: Some Characteristics of the External Ear of American Whites, American Indians, American Negroes, Alaska Eskimos, and Philippinos, Am. J. Anat. **18**:201, 1915.

Becco, R.: Pseudo exostosis del conducto auditivo, Rev. especialid. **4**:580, 1929.

*Becker, F. T.: Congenital Auricular Fistula, Arch. Dermat. & Syph. **48**:520 (Nov.) 1943.

*Becker, O. J.: Surgical Correction of the Abnormally Protruding Ear, Arch. Otolaryng. **50**:541 (Nov.) 1949.

*Becker, S. W., and Brunschwig, A.: Sinus Praeauricularis, Am. J. Surg. **24**:174, 1934.

Belgraver, P.: Over exostoses von de uitwendige gehoorgang, Dissertation, Leyden, 1938.

Bergara, R., and Bergara, C.: Contribución al estudio de las fístulas congénitas del cuello, An. oto-rino-laring. Uruguay **2**:117, 1932.

Berry, G. A.: Note on a Congenital Defect of the Lower Lid, Ophth. Hosp. Rep. London **12**:255, 1888.

Binder: Das Morel'sche Ohr: Eine psychiatrisch-anthropologische Studie, Arch. Psychiat. **20**:514, 1889.

Blake, C. S.: Exostoses in the Ear Canals of Mound Builders, Tr. Am. Otol. Soc. **2**:417, 1880.

——On the Occurrence of Exostoses Within the External Auditory Canal in Prehistoric Man, Am. J. Otol. **2**:81, 1880.

*Bloom, D.: Supernumerary Tragi, Arch. Dermat. & Syph. **36**:884 (Oct.) 1937.

Bol, G., and de Kleyn, A.: Über einen Fall von Polyotie, Acta oto-laryng. **1**:187, 1918-1919.

Brander, T.: Zur Kenntnis der Ätiologie der Ohranhänge, Acta dermat.-venereol. **20**:213, 1939.

*Braun, L.: Studien an Zwillingen: Säuglings- und Kindesalter, Ztschr. Kinderforschung **43**:86, 1934.

Bregeat, P., and Naud, G.: Un nouveau cas de dysostose mandibulo-faciale (syndrome de Franceschetti), Arch. Ophth. **9**:427, 1949.

Breul, F.: Contribution à l'étude des kystes dermoïdes de la région mastoïdienne, Thesis 321, Nancy, 1890.

Brieger, D.: Klinische Beiträge zur Ohrenheilkunde: Mitteilungen aus der Abteilung für Ohrenkranke im Allerheiligen Hospital zu Breslau, Wiesbaden, J. F. Bergmann, 1896, p. 12.

Brohm, F., and Kluska, V.: Mandibulofacial dysostosis (Franceschetti-Zwahlen), Lék. listy **2**:329, 1947.

*Brown, A. M.: Protruding Ears: Plastic Correction, Planning Technique, Operation, Arch. Otolaryng. **47**:809 (June) 1948.

Buchanan, S. A.; Robinson, O. J., and Bryant, D. M.: The Genetics of the Pig, Bibliog. Genet. **12**:1, 1938.

Burkhardt, L., and Burkhardt, I.: Die Gestaltung des äusseren Ohres als konstitutionspathologisches Merkmal, Ztschr. menschl. Vererb.-u. Konstitutions lehre **29**:496, 1949.

Burton, F.: Aural Exostosis, Ann. Otol. Rhinol. & Laryngol. **32**:97, 1923.

Caldera, C.: Nuova varietà di fistula auris congenita e contemporaneo sdoppiamento del condotto uditivo esterno, Arch. ital. otol. **33**:55, 1922.

Carp, L., and Stout, A. P.: Branchial Anomalies and Neoplasms, Ann. Surg. **87**:186, 1928.

Carrière, R.: Über erbliche Ohrformen, insbesondere das angewachsene Ohrlaeppchen, Ztschr. induktive Abstammungs u. Vererbungslehre **28**:238, 1922.

Castle, W. E., and Reed, S. C.: Studies of Inheritance in Lop-Eared Rabbits, Genetics **21**:297, 1936.

Cauchoix, A.: Les fistules congénitales du cou de variété sus hyoidienne à propos d'un cas personnel, Bull. et mém. Soc. nat. de chir. **51**:111, 1925.

Chiari, H.: Vollständiger kongenitaler Defekt der linken Concha auriculae, Verhandl. path. Gesellsch. Dresden **11**:247, 1907.

Chimani, F.: Aneurysma cirsoideum aus der Ohrmuschel, Arch. Ohrenh. **8**:64, 1874.

Cini, C.: Quiste dermoideo de la región mastoidea, Semana méd. **1**:862, 1930.

Citelli, S., cited by Joseph, L.: Arch. Ohrenh. **93**:290, 1914.

Clara, M.: Die arterio-venösen Anastomosen, Leipzig, Johann Ambrosius Barth, 1939.

*Cockayne, E. A.: Inherited Abnormalities of the Skin and Its Appendages, London, Oxford University Press, 1933.

*Coe, H. E.: Congenital Deformities of External Ear, Northwest Med. **38**:135, 1939.

Collins, E. T.: Symmetrical Congenital Notches in Outer Part of Each Lower Lid and Defective Development of the Malar Bones, Tr. Ophth. Soc. U. Kingdom **20**:90, 1900.

Colomiatti, G.: Sopra i vizi di conformazione dell'orecchio esterno, Indipendente di Torino **4**:8, 1873.

Comes, S.: Su una ciste della regione mastoidea, Arch. ital. otol. **45**:559, 1934.

Congdon, E. D.; Rowhanavongse, S., and Varamisara, P.: Human Congenital Auricular and Juxta-Auricular Fossae, Sinuses and Scars (Including the So-Called Aural and Auricular Fistulae) and the Bearing of Their Anatomy upon the Theories of Their Genesis, Am. J. Anat. **51**:439, 1932.

Connon, F. E.: Inheritance of Ear Pits in 6 Generations of a Family, J. Hered. **32**:413, 1941.

Costello, M. J., and Shephard, J. H.: Supernumerary External Ears, Arch. Otolaryng. **29**:695 (April) 1939.

*Dahlberg, G.: Twin Births and Twins from a Hereditary Point of View, Stockholm, A. B. Tiden, 1926.

Davis, O. C. M., and Phillips, P.: Some Observations on Physical Stigmata, Clin. J. **66**:29, 1937.

Debusmann: Familiäre kombinierte Gesichtsmissbildung im Bereich des ersten Viszeralbogens, Arch. Kinderh. **120**:133, 1940.

Desfosses, P.: Les oreilles, Presse méd. **38**:681, 1930.

Dingman, R. O.: Congenital Preauricular Sinus, Arch. Otolaryng. **29**:982 (June) 1939.

*Dragisic, B.: Angeborene Ohrfisteln und ihre differential-diagnostische Bedeutung im Kindesalter, Wien. med. Wchnschr. **86**:748, 1936.

Druss, J. G., and Allen, B.: Congenital Fistula of the Neck Communicating with the Middle Ear, Arch. Otolaryng. **31**:437 (March) 1940.

Dunlop, G., and Hammond, J.: The Growth and Proportions of the Rabbit's Ear in Relation to Body Weight, J. Genetics **34**:463, 1937.

Edmonds, H. W., and Keeler, C. E.: Natural "Ear-Ring" Holes: Inherited Sinuses of Ear Lobes, J. Hered. **31**:507, 1940.

Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose, and Throat, Baltimore, Williams & Wilkins Company, 1947.

Ertl, E.: Glomusgeschwulst der Ohrmuschel, Monatsschr. Ohrenh. **77**:15, 1943.

*Ewing, M. R.: Congenital Sinuses of the External Ear, J. Laryng. & Otol. **61**:18, 1946.

Faleeva, V. L.: Case of Pulsating Angioma Arteriale Racemosum of the Auricle, Zhur. ush. nos. i. gorl. bolez. **16**:264, 1939.

Fano, S.: Observations de kyste congénitale de la région occipito-mastoidienne gauche, Union méd. Paris **13**:42, 1872.

*Frederici, F.: De l'interpretation embryologique de la fistula preauricularis congenita, Acta oto-laryng. **14**:532, 1930.

Feldman, H. W.: The Genetics of the Ear of the House Mouse, Proc. 6th Internat. Cong. Genetics **2**:51, 1932.

Féré, C., and Séglas, J.: Contribution à l'étude de quelques variétés morphologiques du pavillon de l'oreille humaine, Rev. anthropol. **15**:226, 1886.

Ferreri, G.: Esostosi del condotto uditive esterno, Arch. ital. otol. **15**:353, 1904.

*Fischer, H.: Les dysmorphies congénitales craniofaciales et leurs syndromes cliniques, Paris, Vigot Frères, 1929.

Flint, C. P.: Sinus of First Branchial Cleft, Ann. Surg. **48**:165, 1908.

*Foucar, H. O.: Congenital Abnormalities of External Ear, Canad. M. A. J. **43**:26, 1940.

Fournier, G.: A propos d'un cas de fistule congénitale de l'oreille, Rev. laryng. **40**:145, 1919.

Fowler, E. P., Jr., and Osmun, P. M.: New Bone Growth Due to Cold Water in the Ears, Arch. Otolaryng. **36**:455 (Oct.) 1942.

Fox, M. S.: Congenital Aural Fistula, ibid. **35**:431 (March) 1942.

Franceschetti, A., and Klein, D.: The Mandibulo-Facial Dysostosis: A New Hereditary Syndrome, Acta ophth. **27**:143, 1949.

——and Valerio, M.: Malformations associées des yeux et des oreilles, Confinia neurol. **6**:255-257, 1945.

——and Zwahlen, P.: Un syndrome nouveau: La dysostrose mandibulo-faciale, Bull. acad. suisse sc. med. **1**:60, 1944.

Gates, R. R.: Human Genetics, New York, The Macmillan Company, 1946.

Gaus, W.: Zur Frage der Vererbung von Missbildungen des äusseren Ohres, Erbbl. Hals-Nasen- u. Ohrenärzt, Nos. 1-2, pp. 20-30, March, 1939; in Hals-Nasen- u. Ohrenarzt (Pt. 1) **30**: March, 1939.

Gayral; Bru, and Paintandre: Dysostose mandibulo-faciale (syndrome de Franceschetti), J. radiol. et électrol. **31**:97, 1950.

*Geipel, G.: Ein Winkelmesser für den Stellungswinkel des menschlichen Ohres, Ztschr. Morph. u. Anthropol. **32**:403, 1933.

Genetic Studies with Swine at the Idaho Station, Bulletin 179, Idaho Agricultural experiment station, 1931, p. 20.

German, T.: Retroauriculäre Dermoidzyste, Ztschr. Laryngol., Rhinol. **15**:368, 1927.

*Geschelin, I.: Ein Fall von angeborener Missbildung in der Ohrmuschelgegend, Arch. Ohren-, Nasen- u. Kehlkopfh. **136**:332, 1933.

Geyer, E.: Vererbungsstudien am menschlichen Ohre, Mitteil. anthropol. Gesellsch. Wein. **58**:6, 1928.

Probleme der Familienanthropologie, ibid. **64**:295, 1934.

Studien am menschlichen Ohre: Stellung und Faltung der Ohrmuschel, Anthropol. Anz. **13**:101, 1936.

van Gilse, P. H. G.: Die unbedeutende Rolle der Konstitution gegenüber dem Kaltwasser-Reiz für das Entstehen der Gehörgangsexostosen, Monatsschr. Ohrenh. **711**:935, 1937.

Des observations ultérieures sur la genèse des exostoses du conduit externe par l'irritation d'eau froide, Acta oto-laryng. **26**:343, 1938.

*——Hinnen, A. B., and Nieuwenhuyse, A. C.: Heredity in the Field of Otolaryngology, Bibliog. Genetica **13**:301, 1942.

Goldmann, E.: Eine ölhaltige Dermoidcyste mit Riesenzellen, Zeiglers Beitr. path. Anat. **7**:555, 1890.

*Gradenigo, G.: Beitrag zur Morphologie der Anthelix der menschlichen Ohrmuschel, Ztschr. Ohrenh. **21**:289, 1891.

Die Missbildungen der Ohrmuschel, Arch. Ohrenh. **34**:281, 1893.

Greene, H. S., and Saxton, J. A., Jr.: Hereditary Brachiodactylia and Allied Abnormalities in the Rabbit, J. Exper. Med. **69**:301, 1939.

Gritti, P.: Cisti dermoide del solco retroauricolare destro, Arch. ital. otol. **43**:277, 1932.

Gruber, J.: Coloboma auriculae, gelungene Plastik, Monatsschr. Ohrenh. **30**:63, 1896; Lehrbuch der Ohrenheilkunde, ed. 2, Vienna, C. Gerolds Sohn, 1888.

Grüneberg, H.: The Genetics of the Mouse, London, Cambridge University Press, 1943.

Guild, S. R.: Histologic Otosclerosis, Ann. Otol., Rhin. & Laryng. **53**:246, 1944.

Guranowski, L.: Ein Fall von Duplizität des äusseren Gehörganges, Ztschr. Ohrenh. **34**:245, 1899.

*Guszman, J.: Beiträge zur Lehre der branchiogenen Ohr und Halsanhänge, Ztschr. ges. Anat. **81** (pt. 1):554, 1926.

Gutierrez, A., and Montserrat, J. L.: Aneurisma cirsoideo de la región auricular derecha y adyacencias, Bol. y trab. Soc. cir. Buenos Aires **17**:504, 1933.

Guyot, R.: Les fistules auriculaires, Ann. oto-laryng., p. 1227, Dec., 1934.

Habermann, J.: Über Verdoppelung des äusseren Gehörganges, Arch. Ohrenh. **50**:102, 1900.

Hager, A.: Epidermoid-Cyste der Ohrmuschel, Monatsschr. Ohrenh. **84**:106, 1950.

Haike, H., in Brugsch, T., and Lewy, F. H.: Die Biologie der Person, Vol. 3, Berlin, Urban & Schwarzenberg, 1930, p. 577.

Halász, H.: Dermoidcyste an der Mastoidealgegend, Arch. Ohrenh. **63**:141, 1904.

Hanada, K.: Taschenohr (Kubo) und Vererbung, Jap. J. M. Sc., Pt. XII Oto-Rhino-Laryng. **2**, Abstract 54, 1938.

Harding, H.: Ein Beitrag zur Kenntnis der kongenitalen Halsfisteln, Dissertation, Kiel, 1890.

Havens, F. Z.: Congenital Branchiogenic Preauricular Sinus: A Note Regarding Its Treatment, Arch. Otolaryngol. **29**:985 (June) 1939.

Heck, K.: Über ein Choristom der Wartzenfortsatzgegend, Beitr. prakt. u. theoret. Hals-, Nasen- u. Ohrenh. **31**:426, 1935.

Hermans, R.: Case Presented on Coloboma, Bull. Soc. belge ophth., **73**:60, 1936.

Heusinger: Hals-Kiemen fisteln von noch nicht beobachteter Form, Arch. path. Anat. **29**:358, 1864.

Hilden, K.: Über die Form des Ohrläppchens beim Menschen und ihre Abhänigheit von Erbanlagen, Hereditas **4**:351, 1923.

——Studien über des Vorkommen der Darwinschen Ohrspitze in der Bevölkerung Finnlands, Fennia **52**:39, 1929.

Hirschfeld, B.: Fistel am Ohr, Berl. klin. Wchnschr. **45**:517, 1908.

Hlavaček, V.: Exostoses of External Auditory Canal Due to Irritation with Cold Water While Swimming: Affirmation of Gilse's Theory, Časop. lék. česk. **78**:849, 1939.

Hofmann, H.: Degenerationszeichen am Ohr: Eine Untersuchung über die Häufigkeit und den Wert der sogenannten Degenerationszeichen bei Normalen, Verbrechern und Schwachsinnigen, Öffentl. Gesundheits. **6**:573, 1940.

Holm, E.: Et tilfaelde af dysostosis mandibulo-facialis (Franceschetti), Ugesk. læger **48**:1366, 1948.

Hopf, L.: The Human Species Considered from the Standpoint of Comparative Anatomy, Physiology, Pathology and Bacteriology, London, Longmans, Green & Company, 1909.

Horton, B. T., and Hempstead, J.: Congenital Arteriovenous Fistula of Middle Ear and External Auditory Canal, Arch. Otolaryng. **27**:736 (June) 1938.

Hrdlička, A.: Ear exostoses, Smithsonian Miscellaneous Collections **93**:6, 1935.

Hyndman, O. R., and Light, G.: The Branchial Apparatus, Arch. Surg. **19**:410 (Sept.) 1929.

Idaho Agricultural Experiment Station: See Genetic Studies with Swine.

Isakowitz, J.: Eine seltene erbliche Anomalie der Lidspalte (atypisches Lidkolobom?), Klin. Monatbl. Augenh. **78**:509, 1927.

Iwata, H.: Angeborene Missbildung der äusseren Ohres, Beitr. Anat., Physiol., Path. u. Therap. Ohres **5**:258, 1911.

Jacques and Aubriot: Sur un kyste congénital de la région mastoïdienne, Rev. Laryngol. **63**:96, 1922.

Jenkins, R.: Occurrence of a Skin Papillus Through 4 Human Generations, J. Hered. **19**:174, 1928.

Jenks, A. E.: Pitted Ear Lobes of Congenital Origin, ibid. **7**:553, 1916.

Johnston, R. H.: A Large Dermoid Tumor of the Mastoids, J. Eye, Ear, & Throat Dis. **8**:124, 1903.

Johnstone, I. L.: A Case of Deficiency of the Malar Bones, with Defect of the Lower Lids, Brit. J. Ophth. **27**:21, 1943.

Jones, F. W., and Wen, I. C.: Development of External Ear, J. Anat. **68**:525, 1934.

*Josa, L.: Fistulas of First Branchial Arch, Orvosi hetil. **80**:973, 1937.

Joseph, L.: Beitrag zur Lehre von den kongenitalen Geschwülsten der Ohrmuschel, Arch. Ohrenh. **93**:290, 1914.

Kahler, O.: Zur Frage der Polyotie, Arch. Ohren-, Nasen-, u. Kehlkopfh. **142**:248, 1936.

Kaplan, S., and Donlon, J. J.: Bony Dermoid Cyst of the External Auditory Meatus Resembling Deciduous Tooth, Arch. Otolaryng. **34**:354 (Aug.) 1941.

*Karutz: Studien über die Form des Ohres, Ztschr. Ohrenh. **30**:242, 1897; **30**:344, 1897; **31**:11, 1897.

Kaufmann, D.: Über doppelseitige Missbildungen des Gehörorganes, ibid. **39**:210, 1901.

Keith, A.: Three Demonstrations on Congenital Malformations of Palate, Face, and Neck, Brit. M. J. **2**:438, 1909.

Kepes, P.: Hämangiome des Ohres, Monatsschr. Ohrenh. **72**:795, 1938.

Kessel, O. G.: Exostosenstammbaum, Ztschr. Hals-, Nasen-, u. Ohrenh. **8**:266, 1924.

Kilner, T. P.: Notes to the Case Described by I. Mann, Brit. J. Ophth. **27**:18, 1943.

Kindred, J. E.: Inheritance of a Pit in the Skin of the Left Ear, J. Hered. **12**:366, 1921.

*Klaber, R.: Two Cases of Pre-auricular Fistula, Proc. Roy. Soc. Med. **28**:1553, 1935.

Kobozieff, N., and Pomriaskinsky-Kobozieff, N. A.: Sur des nouvelles anomalies héréditaires du pavillon de l'oreille chez la souris, Bull. Mus. nat. hist. naturelle (Series 2) **9**:44, 1937.

Sur les anomalies héréditaires de l'oreille ext. chez la souris, Compt. rend. Soc. biol. **133**:386 1940.

Nouvelles recherches sur la mode de transmission héréditaire des anomalies des oreilles chez la souris, Proc. Seventh Internat. Cong. Genetics, Stockholm, 1941, p. 177.

Sur l'anomalie de l'oreille externe chez la souris: Diversité phénotypique et fréquence, Compt. rend. Soc. biol. **139**:243, 1945.

Sur l'anomalie de l'oreille externe chez la souris: Transmission héréditaire des formes uni- et bilatérales, ibid. **139**:245, 1945.

Anomalie de l'oreille externe chez la souris: Sur la constitution genotypique des souris atteintes d'anomalie faible (abaissement du pavillon) et d'anomalie forte (pavillon tronqué), ibid. **139**:305, 1945.

König, F.: Über fistula colli congenita, Arch. klin. Chir. **51**:578, 1895.

Krampitz, P.: Über einige seltene Formen von Missbildungen des Gehörgorgans, Ztschr. Ohrenh. **65**:44, 1912.

*Krepuska, S.: Oto-laryngologische Untersuchungsresultate bei Zwillingen, Ztschr. Hals-, Nasen- u. Ohrenh. **42**:345, 1937.

Kubo, I.: Über das Taschenohr (Kubo) und die plastische Operation dieser Missbildung, Jap. J. M. Sc., Pt. XII, Oto-Rhino-Laryngol. **2**, Abstract 55, 1938.

Küttner, H.: Die Hyomandibular-Fistel: Eine neue Form der angeborenen Halsfistel, Deutsche med. Wchnschr. **39**:489, 1913.

Landauer, M.: Ein Beitrag zur Kasuistik der kongenitalen Missbildungen des Gehörorganes, Inaugural Dissertation, Munich, 1898.

Langer, C.: Über die Form- und Lageverhältnisse des Ohres, Mitt. anthropol. Gesellsch. Wien. **2**:115, 1882.

Leicher, H.: Die Vererbung anatomischer Variationen der Nase, ihrer Nebenhöhlen und des Gehörorgans, Munich, J. F. Bergmann, 1928.

Leisse, O.: Fisteln im Ohrgebiet, Hals- Nasen- Ohrenarzt, Berlin **1**:275, 1949.

Lenzi, L.: Aneurisma cirsoideo del pabellón auricular, Bol. y trab. Soc. cir. Buenos Aires **16**:349, 1932.

Leopold, I. H.; Mahoney, J. F., and Price, M. L.: Symmetric Defects in the Lower lids Associated with Abnormalities of the Zygomatic Processes of the Temporal Bones, Arch. Ophth. **34**:210 (Sept.) 1945.

Levesque, L.: Contribution à l'étude des kystes dermoïdes de l'apophyse mastoïde et du pavillon de l'oreille, Thesis, Paris, 1907.

Levinstein, O.: Unvollständige innere Hals-Kiemenfistel in Verbindung mit doppelseitiger Fistula præauricularis congenita, Arch. Laryng. u. Rhinol. **23**:128, 1910.

Linder, L.: The Angle of the External Ear to the Median Plane of the Skull in Children, Acta genet. et statist. med. Basel **1** (No. 1): 92, 1948.

van Lint, A., and Hennebert, P.: Atrophie (Colobome) des paupières inférieures, des oreilles et du maxillaire inférieur, Bruxelles-méd. **17**:1065, 1937.

Loebell, H., and Nauck, E.: Die funktionelle Anatomie des menschlichen Felsenbeines, Ztschr. Hals-, Nasen- u. Ohrenh. **34**:129, 1933.

Lönnberg, E.: Missbildningar, Iakttagna hos däggdjur, Fauna och Flora 1930, p. 49.

Looser, E.: Über Spätrachitis und Osteomalazie, Deutsche Ztschr. Chir. **152**:210, 1920.
Über Zysten und braune Tumoren, ibid. **189**:113, 1924.
Zur Pathogenese der Ostitis fibrosa, Verhandl. deutsche path. Gesellsch. **21**:91, 1926.

Luke, C. J.: Congenital Arteriovenous Fistulae of Pinna, Canad. M. A. J. **46**:167, 1942.

Lüscher, E.: Isoliertes Chondrom im knöchernen Teil des äusseren Gehörorgans ("Auricularanhang"?), Arch. Ohren-, Nasen u. Kehlkopfh. **129**:63, 1931.

Lush, J. L.: An Hereditary Notch in the Ears of Jersey Cattle, J. Hered. **13**:8, 1922.
"Double Ears" in Brahma Cattle, ibid. **15**:93, 1924.
Earlessness in Karakul Sheep, ibid. **21**:112, 1931.

Lynch, C. J.: Short Ears, an Anatomical Mutation in the House Mouse, Am. Natural. **55**:421, 1922.

Lynch, R. C.: Congenital Absence of Both Ears, Laryngoscope **23**:1050, 1913.

Mac Collum, D. W.: The Lop Ear, J. A. M. A. **110**:1427 (April 30) 1938.

Maggiorotti, U.: L'amartoma racemoso del padiglione, Arch. ital. laring. (Supp.) **51**:117, 1932.

*Magnotti, T.: Sulle malformazioni congenite dell'orecchio esterno, Arch. ital. otol. **41**:303, 1930.

Mann, I.: Deficiency of the Malar Bones with Defect of the Lower Lids, Brit. J. Ophth. **27**:13, 1943.

Marshall, G. G.: Dermoid Teeth in the External Auditory Canal, New England J. Med. **214**:202 (Jan. 30) 1936.

*Martilotti, F.: Sulle malformazioni congenite dell'orecchio esterno, Pediatria **45**:337, 1937.

*Marx, H.: Die Geschwülste des Ohres, Handb. spez. path. Anat. u. Histol. **12**:490, 1926.
Die Missbildungen des Ohres, ibid. **12**:609, 1926.

Mayer, O.: Bericht über die Ergebnisse weiterer Untersuchungen zur Otosklerosefrage, Ztschr. Hals-, Nasen- u. Ohrenh. **6**:280, 1923.
Die Ursache der Knochenbildung bei der Otosklerose, Acta oto-laryngol. **15**:35, 1931.
Über die Entstehung der Spontanfrakturen der Labyrinthkapsel und ihre Bedeutung für die Otosklerose, Ztschr. Hals-, Nasen- u. Ohrenh. **26**:26, 1930.
Referat über die pathologische Anatomie und Pathogenese der Otosklerose, Second Congrès internat. d'oto-rhino-laryngologie, Blass, Madrid, 1932, Vol. 1, p. 8.
Die Entstehung der Exostosen des äusseren Gehörganges, Ztschr. Laryng. Rhin. Otol. **28**:565, 1949.

McDonough, M. S.: On Inheritance of Ear Pit (Fistula Auris Congenita) with Special Reference to Twins, J. Hered. **32**:169, 1941.

McEnery, E. T., and Brennemann, J.: Multiple Facial Anomalies, J. Pediat. **11**:468, 1937.

McPheters, B. W., and Little, C. C.: Hound Eared Mice, J. Hered. **24**:157, 1933.

Mengele, J.: Zur Vererbung der Ohrfisteln, Erbarzt **8**:59, 1940.

Meyer, H.: Über den Einfluss des Badens aur die Exostosenbildung des äusseren Gehörganges, Dissertation, Marburg, 1948.

Meyer, W.: Eine seltene Gehörsmissbildung beim Schwein, Berlin. Tierärtzl. Wchnschr. **26**:613, 1910.

*Meyer, W. H.: Congenital Cysts and Fistulas of the Neck, Ann. Surg. **95**:13, 1932.

Miller, J. B., and Moore, P. M.: Fistula Auris Congenita, Arch. Otolaryng. **51**:245 (Feb.) 1950.

Miyake, S.: On the Auricular Exostosis of the Neolithic People from Shell Mounds of Ota, Bingo, Jap. J. M. Sc., Anatomy **4**:25, 1934.

Mohr, C. L.: Letalfaktoren bei Haustieren, Züchtungskunde **4**:105, 1929.

Moldenhauer, W.: Die Missbildungen des Ohres, in Schwartze, V. A.: Handbuch der Ohrenheilkunde, Leipzig, 1892, Vol. 1, p. 154.

Möller-Holst, G.: Ergebnisse einer Forschungsreise nach Chile-Bolivien: Der äussere knocherne Gehörgang südmerikanischer Schädel, Ztschr. Morphol. & Anthrop. **31**:63, 1932.

Montgomery, M. L.: Congenital Auricular Fistula: Report of 3 Cases in the Same Family, S. Clin. North America **11**:141, 1931.

Moraza, M.: Dos casos de gigantismo parcial, Med. ibera **2**:630, 1930.

Nager, F.: Über mediane Nasenfisteln, Monatsschr. Ohrenh. **66**:60, 1932.

Navis, H.: Dysostosis mandibulo-facialis, Nederl. tijdschr. geneesk **92**:740 (March 6) 1948.

Noguchi, M., and Kotake, Y.: Über das Taschenohr (Kubo), Oto-rhino-laryng. **9**:820, 1936.

Nordby, J.: Congenital Skin, Ear, and Skull Defects in a Pig, Anat. Rec. **42**:267, 1929.
Congenital Ear and Skull Defects in Swine, J. Hered. **21**:499, 1930.

*O'Donnel, J. M.: Congenital Preauricular Sinuses: 3 Cases, M. J. Australia **1**:405, 1948.

Onodi, S.: Über kongenitale Ohrenfisteln, Arch. Ohrenh. **102**:128, 1918.

Oppikofer, E. K.: Hals-Ohrfistel (Fistula collo-auralis congenita) und Trommelfellmissbildung, Prac. oto-rhino-laryng. **8**:515, 1946.

Ostmann, P.: Beiträge zu dem Vorkommen von Exostosen des äusseren knöcheren Gehörganges bei den verschiedenen Völkerrassen, Monatsschr. Ohrenh. **28**:237, 1894.

*Otermin Aguirre, J. A.: Handle-Shaped Ears, J. M. Buenos Aires **2**:347, 1948.

Otsuka, H.: Über das Taschenohr (Kubo), Oto-rhino-laryng. **9**:813, 1936.

Pastore, P., and Erich, J.: Congenital Preauricular Cysts and Fistulas, Arch. Otolaryng. **36**:120 (July) 1942.

Penick, R. M., Jr.: Preauricular Sinuses: Diagnosis and Treatment, South. M. J. **38**:103, 1945.

Pipkin, A. C., and Pipkin, S. B.: Ear Pits and Albinism in a Negro Family, J. Hered. **34**:242, 1943.

Pires de Lima, J. A.: Dysmorphie des oreilles, Ann. anat. path. **7**:377, 1930.

——and Monteiro, H. B.: Aparelho branguinal e suas perturbações evolutivas, Arq. anat. antrop. **8**:185, 1923.

Pirodda, E.: Note clinico terapeutiche su un caso di angioma arteriale racemoso del padiglione auricolare, Oto-rino-laring. ital. **15**:287, 1947.

Podgaetsky, B.: Case of Unilateral Gigantic Development of Auricle, Zhur. ush. nos. igorl. bolez. **15**:551, 1938.

Potter, E. L.: A Hereditary Ear Malformation Transmitted Through 5 Generations, J. Hered. **28**:255, 1937.

Powell, E. F., and Whitney, D. D.: Earlobe Inheritance: Unusual 3-Generations Photographic Pedigree Chart, J. Hered. **28**:185, 1937.

Quelprud, T.: Untersuchungen der Ohrmuschel von Zwillingen, Ztschr. induktive Abstammungs- u. Vererbungslehre **62**:160, 1932.
Zur Erblichkeit des Darwinschen Höckerchens, Ztschr. Morphol. u. Anthrop. **34**:343, 1934.
On Inheritance of Darwin's Tubercle, Eugenical News **20**:3 (Feb. 25) 1935.
Familienforschungen über Merkmale des äusseren Ohres, Ztschr. induktive Abstammungs- u. Vererbungslehre **67**:296, 1935.

Die Ohrmuschel und ihre Bedeutung für die erbbiologische Abstammungsprüfung, Erbarzt **1** and **2**:421, 1935.

Die Vererbung des crus cymbae der menschlichen Ohrmuschel, Proc. Anthropol. Cong., Copenhagen, 1939, p. 192.

Ear Pit and Its Inheritance: Fistula Auris Congenita, Described in 1864, Still Genetical and Embryological Puzzle, J. Hered. **31**:379, 1940.

Variability and Genetics of Human External Ear, Proc. Internat. Cong. Genetics (1939) **7**:243, 1941.

Regmunt-Sobieszczanski, L.: New Approach to the Tubercle of Darwin, Folia Morphol. **4**:85, 1933.

Reh, H.: Über einen Fall von angeborenen Fisteln der Augen-Nasenrinne, kombiniert mit angeborenen Ohrfisteln und Anosmie, Klin. Monatsbl. Augenh. **104**:55, 1940.

*Richer, P.: Nouvelle anatomie artistique du corps humain: Part II. Morphologie, la femme, Paris, Libraire Plon, 1928.

Riddell, W. J. B.: Studies in Classification of Eye Colour, Ann. Eugenics **11**:235, 1942.

Ride, L., in discussion, Tr. 11th Assembly of the Internation Federation of Eugenic Organizations, Arch. Internat. Klaus-Stiftg. **10**:42, 1935.

Riedel: Über die ins Mittelohr führende Kiemengangsfistel, Verhandl. deutsch. Gesellsch. Chir. **26**:125, 1897.

Ritzmann, E. G.: Breeding Earless Sheep, J. Hered. **11**:238, 1920.

Mendelism of Short Ears in Sheep, J. Agricult. Res. **6**:797, 1916.

Roques-Sativo, R.: Dysplasies et tumeurs du pavillon de l'oreille, Rev. laryng. **57**:304, 1936.

Ruttin, E.: Zur Frage der Fistula auris congenita und der Auricularanhänge, Wien. med. Wchnschr. **77**:1019, 1927.

Über Exostosen und Hyperostosen des äusseren Gehörgangs, Acta oto-laryng. **18**:381, 1932-1933.

in discussion on Šercer, A.: Ibid. **20**:59, 1934.

De Ruyter, cited by Riedel: Verhandl. deutsch. Gesellsch. Chir. **26**:125, 1897.

Ruzic, J.: Der siebente Fall kompletter Anotie, Acta oto-laryng. **36**:186, 1948.

Sannicandro, G.: Tumore glomico dell'orecchio con particolari aspetti istologici, Dermosifilografo **11**:424, 1936.

Sanvenero-Roselli, G.: Die angeborenen Gesichts- und Kiefermissbildungen, Plast. chir. Milan **1**:184, 1940; Deutsch. Zahnärztl. Ztschr. **3**:816, 1948.

*Sawatari, J., and Hama, T.: Über Exostosen des äusseren Gehörgangs an den Aino-Schädeln, Ztschr. Oto-Rhino- u. Laryngol. **41**:101, 1935.

Über Exostosen am äusseren Gehörgang des Aino-Schädels, Jap. J. M. Sc., Anatomy **6**:179, 1937.

Scalori, G.: Considerazioni sopra un caso di fistola congenitale del lobulo auricolare, Valsalva **11**:606, 1935.

Schachter, M.: Atresie congénitale des paupières inférieures, faciès de "Clown" et malformations sequelettiques multiples chez un petit oligophrène: Malformations identiques chez le père, Ann. paediat. **169**:345, 1947.

Recherches sur les fossettes para-auriculaires, ou fistules auriculaires congénitales (fistula auris congenita), Schweiz. med. Wchnschr. **79**:343, 1949.

Schofield, R.: Inheritance of a Bilobed Ear, J. Hered. **3**:517, 1917.

Schüller, J.: Zur Vererbung der Fistula auris bzw. auriculae congenita: Zugleich ein klinischer Beitrag, München. med. Wchnschr. **76**:160, 1929.

Schwalbe, G.: Beiträge zur Anthropologie des Ohres, Internat. Beitr. wissensch. med. Virchow Festschr. **1**:95, 1891.

Das äussere Ohr, in Handbuch der Anatomie des Menschen, Jena, G. Fischer, 1897, Vol. 5, Pt. 2, p. 125.

*Beiträge zur Kenntnis des äusseren Ohres der Primaten, Ztschr. Morphol. u. Anthropol. **19**:545, 1916.

*Schwendt, A.: Über congenitale Missbildungen des Gehörorgans in Verbindung mit branchiogenen Cysten und Fisteln, Arch. Ohrenh. **32**:37, 1891.

Seebohm, E.: Über einen seltenen Fall einer Fistula auris et colli congenita, Diss., Berlin, 1938.

Seifert, E.: Über die sogenannte Fistula auris congenita, Deutsch. Ztschr. Chir. **20**:118, 1928.

Seligmann: Über Exostosen an Peruanerschädeln, Sitzungsb. d. k. Akad. Wissensch. Wien. 1864, p. 55.

Selkirk, T. K.: Fistula Auris Congenita, Am. J. Dis. Child. **49**:431 (Feb.) 1935.

Šercer, A.: Komplette Anotie kombiniert mit Aplasie der Tonsille, Ztschr. Hals-, Nasen- u. Ohrenh. **22**:75, 1928.

Beitrag zur Kenntnis der Formanomalien des äusseren Ohres, Acta oto-laryng. **20**:59, 1934.

Ptosis auriculae congenita, Arch. Ohren-, Nasen- u. Kehlkopfh. **149**:298, 1941.

Simpson, W. L.: Unusual Ear Cases: Salivary Fistula of Mastoid Region, Bilateral Cysts of Auditory Meatus, South. M. J. **22**:215, 1929.

Sington: Über einen Fall von sogenannter Ohrfistel eines Pferdes, Ztschr. Veterinärk. **39**:356, 1927.

Smith, F.: Congenital Arteriovenous Fistula in the Tympanum, Arch. Otolaryng. **10**:32 (July) 1929.

Snyder, L. H.: The Principles of Heredity, ed. 3, Boston, D. C. Heath & Co., 1946.

Spira, R.: Seltener Fall einer kombinierten angeborenen Missbildung des äusseren Gehörgangs, Monatsschr. Ohrenh. **41**:664, 1907.

Starkenstein, E.: Über die Vererbung einer branchiogenen Fistel, Med. Klin. **24**:701, 1928.

*Steiner, A.: Zwei Fälle von Fistulae auris congenitae, Dermat. Wchnschr. **86**:325, 1928.

Stewart, T. D.: The Tympanic Plate and External Auditory Meatus in the Eskimo, Am. J. Phys. Anthropol. **17**:481, 1933.

Stiles, K. A.: The Inheritance of Pitted Ear, Genetics **26**:171, 1941; J. Hered. **36**:53, 1945.

Straith, C. L., and Lewis, J. R.: Associated Congenital Defects of Ears, Eyelids and Malar Bones (Treacher Collins Syndrome), Plast. & Reconstruct. Surg. **4**:204, 1949.

*Suarez, C.: Un caso de duplicidad-unilateral del tímpano, An. Casa salud Valdecilla **10**:63, 1947.

Sutton, J. B.: On Branchial Fistulae, Cysts, Diverticula and Supernumerary Auricles, J. Anat. & Physiol. **21**:298, 1887.

*Tildesley, M. L.: Techniques of Ear Length and Breadth Measurement Reviewed and Tested, Arch. Julius Klaus Stiftg. Vererbungsforsch. **24**:337, 1949.

Tommasi, C.: Ipertricosi audiculare famigliare, Arch. Psiquiatr., Neuropat., Antropol., Crim. e. Med. Legale **28**:60, 1907.

*Toynbee, J.: Diseases of the Ear, Philadelphia, Blanchard & Lea, 1860.

*Toyoshima, Y.: Über die Untersuchungsresultate der Fistula auris congenita bei Marinesoldaten, Oto-rhino-laryng. **9**:825, 1936.

Turner, W.: On Exostoses Within the External Auditory Meatus, J. Anat. & Physiol. **13**:200, 1879.

Virchow, R.: Über Missbildungen am Ohr und im Bereiche des ersten Kiemenbogens, Virchows Arch. path. Anat. **30**:221, 1864.

Über krankhaft veränderte Knochen alter Peruaner, Sitzungsb. k. Preuss. Akad. Wissensch., 1885, Pt. 2, p. 1129.

Über einige Merkmale niederer Menschanrassen am Schädel, Physiol. Abhandl. k. Preuss. Akad. Wissensch., 1875, p. 99.

Über Exostosen des Meatus aud., Berlin. Klin. Wchnschr. **30**:635, 1893.

*Vollmer, H.: The Shape of the Ear in Relation to Body Constitution, Arch. Pediat. **54**:574, 1937.

Waardenburg, P. J.: Eine merkwürdige Kombination von angeborenen Missbildungen: Doppelseitiger Hydrophthalmus verbunden mit Akrokephalosyndaktylie, Herzfehler, Pseudohermaphroditismus und anderen Abweichungen, Klin. Monatsbl. Augenh. **92**:29, 1934.

Das menschliche Auge und seine Erbanlagen, Haag, Martinius Nijhoff, 1932, pp. 50-51.

——and Navis, H.: Beitrag zur Kenntnis der Dysostosis mandibulafacialis und ihrer Ontogenese, Acta genet. et statist. med. **1**:219, 1949.

Walde, I.: Zur Frage der Gehörorgansverdoppelung, Ztschr. Hals-, Nasen- u. Ohrenh. **32**:227, 1938.

Wassin, B.: Ohrlosigkeit bei Schafen und Zeigen, Ztschr. induktive Abstammungs- u. Vererbungslehre **49**:95, 1928.

*Watson-Williams, E.: Congenital Abnormalities of External Ear, Bristol, Med.-Chir. J. **48**:273, 1931.

Weber, W.: Über Art, Häufigkeit und Genfrequenz der Missbildungen unserer Haustiere, nebst einem Fall von Agenesie des Geruchsapparates bei einem Kalb, Schweiz. Arch. Tierh. **88**:497, 1946.

Welcker, H.: Über knöcherne Verengung und Verschliessung des äusseren Gehörorganes, Arch. Ohrenh. **1**:163, 1864.

Wenglowski, R.: Über die Halsfisteln und Cysten, Arch. klin. Chir. **100**:789, 1913.

Whitney, D. D.: Three Generations of Ear Pits, J. Hered. **30**:323, 1939.

van den Wildenberg: Fistules congénitales du cou, Ann. mal. oreilles **45**:677, 1926.
Les exostoses profondes du conduit auditif, Acta oto-rhino-laryng. belg. **1**:173, 1947.

Wildermuth, H. A.: Über Degenerationszeichen bei Epileptischen und Idioten, Med. Cor.-Bl. Württemb. aerztl. Landesv. **56**:313, 1886.

Wreden, R.: Zur Casuistik der angeborenen Missbildungen des Ohres, Monatsschr. Ohrenh. **4**:21, 1870.

Wriedt, C.: Über die Vererbung von Ohrenlänge beim Schaf, Ztschr. induktive Abstammungs u. Vererbungslehre **20**:262, 1919.
Breeding Earless Sheep, J. Hered. **12**:56, 1921.
The Hereditary Relationship of Earlessness and Short Ears in Sheep and the Production of This Type in Norway, ibid. **36**:430, 1925.

Wyman, J.: Report on a Collection of Peruvian Crania, 7th Ann. Rep. Peabody Museum, Cambridge, Mass., 1874, p. 15.

Yamanake, K.: Ein Fall von Taschenohr, Oto-rhino-laryng. (Abstract Sect.) **13**, n.p., (Feb.) 1940.

Yamane, J.: On the Inheritance of an Aural Abnormality in the Ayrshire Cattle, J. Coll. Agric. Tohoku Imp. Univ. **6**:166, 1915, cited by Kobozieff, N., and Pomriaskinsky-Kobozieff, N. A.: Compt. rend. Soc. biol. **133**:386, 1940.

Yokogawa, Y.: Mischgeschwulst des äusseren Gehörgangs, Jap. J. M. Sc., XII. Oto-Rhino-Laryngol. **2**, Abstract 58, 1938.

Young, F.: The Correction of Abnormally Prominent Ears, Surg., Gynec. & Obst. **78**:541, 1944.

Zimmermann: Zur Teratologie des Haustierohres, Arch. wissensch. u. prakt. Tierh. **40**:432, 1914.

Zöllner, F.: Fehldiagnosen, verursacht durch angeborene Fisteln und Cysten an Ohr und Hals, Ztschr. Hals-, Nasen- u. Ohrenh. **32**:54, 1932.

Zwahlen, P.: La dystose mandibulo-faciale, Thesis No. 1826, Geneva, 1944.

"POSTOPERATIVE" HYPERRHINOLALIA (RHINOLALIA APERTA)

EMIL FROESCHELS, M.D.
NEW YORK

HYPERRHINOLALIA means the audible passage of air through the nose at an inappropriate time, namely during the pronunciation of all English vowels and consonants with the exception of m, n, ng, and neighboring vowels. I say they are "audible" because according to Schmidt [1] some air may pass through the nose unperceived by the ear of the listener, an amount so small that it can pass under ordinary pressure an opening of about 3 mm. between the velum and the posterior wall of the pharynx. "Audible" refers to the acoustic acuity of the observer. An ear not too well trained in this respect may not detect small degrees of hyperrhinolalia. Gutzmann's ah-ee test [2] may alleviate the difficulty of diagnosis. Tactile and visual tests are also of some help. If one touches slightly the alae nasi, preferably with the tip of the middle finger, hyperrhinolalia manifests itself by vibrations, especially in pronunciation of vowels. A cold mirror placed horizontally under the nostrils while vowels, syllables, or words, none of which contains a nasal sound, are pronounced will be spotted with moisture if hyperrhinolalia is present. Graphic methods are also available but are too specialized to be used by anyone except a trained phonetician. I doubt that a trained examiner will ever fail to recognize hyperrhinolalia.

It is well known that there are numerous causes of this speech trouble. Here two of them are discussed, namely the organic and the functional insufficiency of the vellum after adenectomy and/or tonsillectomy. It seems to be well established that a postoperative hyperrhinolalia exists, that is to say that a velum organically and functionally perfect before the operation can show signs of weakness afterward. I am not inclined to deny this assumption of postoperative hyperrhinolalia, since sometimes, especially after tonsillectomy, scars can be seen in the pillars and/or in the tissues of the velum. Such scars can prevent the velum from functioning normally. It is also possible that pains after surgery may cause the patient to use unextensive movements of the soft palate and that those weak movements may become habitual. However, I have never seen the development of a "postoperative" hyperrhinolalia when I had an opportunity of examining the patient before the

1. Schmidt, cited by Kaltenborn, A. L.: An X-Ray Study of Velopharyngeal Closure in Nasal and Non-Nasal Speakers, Thesis, Northwestern University, 1948.

2. While the patient says ah-ee repeatedly the examiner closes the patient's nostrils every time during the ee. In hyperrhinolalia the difference between the hyperrhinolalic ah and the "damped" ee will become apparent. There is no such change in the sound in nonhyperrhinolalic people.

operation without finding any hyperrhinolalia. In other words, all my patients who showed a strong hyperrhinolalia after surgery had at least slight signs before operation. Of course there were quite a few cases of anteoperative hyperrhinolalia noticeable to everybody. These are not to be discussed in this article because no one would call this type of peculiar speech "postoperative" only. A case of wrongly assumed postoperative hyperrhinolalia is presented.

A 4½-yr.-old boy was admitted to the Speech and Voice Clinic of Beth David Hospital, New York, April, 1951. He showed a very apparent hyperrhinolalia which his family noticed only after adenotonsillectomy, which was performed in the fall of 1949. Somatic and neurological findings were not significant. The velum did not move during the pronunciation of ah, but the gagging reflex was normal. Schlesinger's test [3] was positive (in supine position hyperrhinolalia diminished because the velum, obeying the law of gravity, sank toward the posterior wall of the pharynx). Therefore the hyperrhinolalia was not of the "active type" (D. Weiss [4]); in other words, the velum was not dragged down by perverse contractions of the velopharyngeal muscles. The mother was asked again whether she was sure that no "nasality" was present before surgery, and she was positive that nobody ever noticed any kind of abnormal speech. But she suddenly remembered that ever since birth at times liquids regurgitated through the patient's nose. That indicates that the velum showed, at least occasionally, signs of inefficiency. It is well known that the function of the velum in swallowing on the one hand and in speaking on the other is neurophysiologically different. Swallowing is a reflex. Even if deliberately performed, the participation of the velum will still be reflexive in nature. In speech, however, the functions of the velum are assumed to belong to the voluntary movements, although these functions do not proceed on a level of distinct consciousness. Experience proves that the speech function of the soft palate may be inadequate while swallowing proceeds normally. The opposite never occurs. If the palate fails to close in swallowing it always also fails in speech. This is the case in bulbar paralysis (fourth ventricle) and in pseudobulbar paralysis (pyramidal tract). The patient's mother reported that after the operation regurgitation stopped. This was to be expected since the boy did not show any neurological findings indicating a defect in the central or peripheral nervous system. Fluoroscopy recently performed by Dr. Brodnitz and me proved that the velum worked normally in swallowing. (To make the velum visible we used the method of Haudek-Froeschels consisting of smearing some barium paste on the velum.)

Why did regurgitation occur anteoperatively? Because one may say the velum was prevented from reaching the posterior wall of the pharynx by the "wedged in" adenoids. However, if the growth hindered the velum from rising why could fluid rise into the nose? Because there exists a certain form of adenoids, according to Gutzmann, which covers only the middle part of the pharyngeal wall, at least in its lower part, so that on both sides of the growth the way to the nose stays open and fluid may regurgitate in this way.[5] Evidently the same condition must have produced hyperrhinolalia, but it was not noticed because no speech expert was consulted.

From that we can conclude that our case is not a case of postoperative hyperrhinolalia, meaning a speech trouble resulting from an operative injury to the velum. Naturally when the adenoids, which acted like a damping cushion on the air entering the nose, were removed the hyperrhinolalia became more conspicuous. So the term "postoperative hyperrhinolalia" seems, in this case, logically unjustified.

---

3. Schlesinger, H.: Case Demonstration, in Wiener Gesellschaft für interne Medzin, Wien. neurol. Zentralbl., 1906.

4. Weiss, D.: Eine noch nichte beschriebene Sprachstörung: Hyperrhinolalia activa, Logopaedie en Phoniatrie, nos. 9 and 10, Oct., 1937.

5. The rhinologist who performed the operation could neither confirm nor deny this assumption.

How can one explain the weakness of the velum? Stein [6] has taught that a muscle meeting too much resistance in its actions may "give up." Since our patient's velum with all probability always met the cushion of adenoids when it intended to rise, functional "paralysis" may have resulted. The boy already showed some improvement when this paper was written after five weeks of treatment with the pushing exercises.[7]

From the observation made in this case and in numerous others of "postoperative" hyperrhinolalia one bit of advice seems to be obviously indicated. Every patient who is being considered for adenectomy or tonsillectomy should first be examined by a logopedist. In this way several inconveniences can be avoided. If insufficient velar function exists (whether or not hyperrhinolalia is noticed by others than the logopedist), the patient or his relatives should be advised that hyperrhinolalia is present and will be more conspicuous after the operation. On this basis the otolaryngologist may reconsider whether or not the operation must be performed for reasons of general health (recurrent inflammation of the tonsils, obstruction of the nose for normal nose breathing, etc.) or of local damage to the ear by adenoids.

SUMMARY

The existence of so-called postoperative hyperrhinolalia can be established scientifically only by logopedic examination of the function of the velum palatinum before adenectomy and/or tonsillectomy. A case history of a boy whose voice had no conspicuous nasality to an untrained person before the operation, but which would doubtlessly have shown a slight hyperrhinolalia and/or an imperfect function of the velum to a logopedist proves the necessity of preoperative logopedic examination. Only in this way can the patient with hyperrhinolalia and/or his relatives be saved from disappointments due to the inevitable consequences of the operation.

133 E. 58th St. (22).

6. Stein, L.: Über die Rhinolalia aperta im Himblick auf die Lehre von der funktionellen Anpassung, Beitr. Anat., Physiol., Path. u. Therap. Ohres. **16**:167-188, 1921.

7. Froeschels, E.: Twentieth Century Speech and Voice Correction, New York Philosophical Library, Inc., 1948, p. 162.

TRACHEOTOMY IN TETANUS

EMANUEL HERZON, M.D.
EDGAR KILLIAN, M.D.
AND
SAMUEL J. PEARLMAN, M.D.
CHICAGO

TYPICAL figures from the recent literature show that the mortality in tetanus varies from 35% [1] to 63%.[2] Experimental physiological [3] and histopathological [4] studies reveal the primary factor to be respiratory failure. In addition, the use of central-nervous-system depressants may contribute to respiratory decompensation.

These facts on mortality, pathogenesis, and causation of death [3] prompted the present inquiry into the rationale of the current treatment of tetanus. In this review six cases of tetanus are presented in which, during the clinical course, tracheotomy was required for the patients' survival.

REPORT OF THE CASES

CASE 1.—F. C., a Negro woman aged 57, entered the hospital on Feb. 7, 1950, at 12:45 a. m., with generalized spasms and trismus of two days' duration. Four days previously she had accidentally spilled boiling water on her left thigh. She applied sodium bicarbonate and soap to the burn. Two days later she was unable to open her mouth or to swallow water, and breathing was difficult. Her neck was stiff, headache was constant, and she felt feverish.

The initial physical examination showed a temperature of 99.6 F., pulse rate of 96, respiration rate of 28, and blood pressure of 140/100. The patient was alert, oriented, and very apprehensive. The significant findings were nuchal rigidity and tenderness, risus sardonicus, and trismus. Through a narrow chink between the teeth, a small laceration of the tongue was seen. The abdominal and back muscles were rigid. On the inner aspect of the left thigh there was a 20 by 16 cm. second degree burn. The patellar reflexes were hypoactive, and the Babinski reflex was negative. Three generalized spasms occurred during the physical examination. The urine was normal. A culture from the burn was later reported as containing Escherichia coli, Proteus morganii, and alpha hemolytic streptococci.

The immediate treatment consisted of 300,000 units of penicillin intramuscularly, sodium amobarbital (sodium amytal®), 7.5 grains (0.5 gm.) intravenously, pentobarbital (nembutal®),

---

From the Ear, Nose, and Throat Department of the Cook County Hospital

Read before a meeting of the Chicago Laryngological and Otological Society, Chicago, Nov. 7, 1950.

1. Schreiber, S. L.: Tetanus in Kanawha Treated in Charleston, West Virginia, Hospital, West Virginia M. J. **43**:398-406 (Dec.) 1947.

2. Huntington, R. W., Jr.; Thompson, W. R., and Gordon, H. H.: The Treatment of Tetanus with Antitoxin, Ann. Surg. **105**:93-96 (Jan.) 1937.

3. Firor, W. M.; Lamont, A., and Shumaker, H. B., Jr.: Studies on the Cause of Death in Tetanus, Ann. Surg. **111**:246-274 (Feb.) 1940.

4. Baker, A. B.: The Central Nervous System in Tetanus, J. Neuropath. & Exper. Neurol. **1**:394-405 (Oct.) 1942.

4.5 grains (0.3 gm.) rectally, and 40,000 units of tetanus antitoxin intramuscularly and 40,000 units intravenously in 10% dextrose. The patient was placed in a quiet room in postural-drainage elevation. Much mucus was aspirated from the nasopharynx.

Two hours after admission, she thrashed about in bed and seized her throat. Respirations became shallow and irregular with noisy forceful expiration. Sodium amobarbital, 7.5 grains (0.5 gm.), was administered intravenously. Two hours later she was comatose with absent corneal reflexes and fixed pupils. Thick, tenacious mucus drooled from her mouth and was aspirated from the nasopharynx. For the next two hours there occurred frequent laryngospasms, lasting two to four respiratory cycles. Chest auscultation revealed coarse rhonchi bilaterally. The temperature rose to 101.4 F. rectally, the respiration rate increased to 48, the pulse rate rose to 106, and the blood pressure dropped to 108/80. The consultant in otolaryngology advised tracheotomy, but the attending physician decided to wait.

Throughout the first hospital day, respirations showed a prolonged expiratory phase with marked suprasternal retraction. Restlessness became so pronounced that restraints were required. During the night, there were brief generalized spasms and mucus was aspirated with a nasopharyngeal catheter. At midnight, 24 hr. after admission, 1.023 gm. of tribromoethanol (avertin®) were given rectally to control the restlessness. The patient became comatose with shallow respiration, and later the respiration consisted only of rapid gasps. Her condition became grave; cyanosis appeared, and respiratory stridor was constant.

At this time, 36 hr. after admission, the temperature was 103.6 F. and the pulse rate 112. The otolaryngological consultants believed that the patient was preterminal, but decided to give her the benefit of the doubt and do a tracheotomy. Thick purulent secretions welled into the tracheotomy wound and were removed by suction. The patient remained in a comatose state, although no more sedation was given. She expired 19 hr. after tracheotomy.

At postmortem examination massive atelectasis of the lungs was revealed. The right upper lobe was slightly crepitant, but the other lobes were deep purple and airless. The entire specimen sank when placed in water. The tracheobronchial tree contained mucopurulent material. The brain weighed 1,150 gm. and showed thickened leptomeninges, flat gyri, and narrow sulci. The vessels were greatly dilated.

We believe that this patient suffered irreversible damage to the respiratory system due to the delay in performing tracheotomy.

CASE 2.—M. P., a white man aged 45, was admitted to the hospital on Jan. 4, 1950, complaining of numbness of the feet for some hours. He had slept outdoors the night before and was suffering from exposure. The numbness was associated with a burning sensation which had become progressively worse.

On examination, the temperature was found to be 98.8 F., the pulse rate 88, the respiration rate 20, and the blood pressure 124/80. There were scattered edematous blebs over the dorsa of the feet and bluish discoloration of all the toes. The laboratory findings revealed nothing significant. The diagnosis was severe frostbite of both feet.

The immediate treatment consisted of a 10 min. scrubbing with soap and water with sterile precautions, followed by a petrolatum gauze and pressure dressing. Penicillin, in doses of 300,000 units, was given four times a day. From the third to the fifth hospital day, the patient developed but recovered from a slight cellulitis of the areas. By the seventh day, a gangrenous demarcation line appeared on the last three toes of the right foot.

On the 17th hospital day, a superficial débridement of the right foot was carried out with sterile precautions. That evening, the patient had a slight trismus which increased within 24 hr., and he had difficulty in swallowing sips of water. Opisthotonos was present, but the sensorium was clear. A diagnosis of tetanus was made, and after skin tests with tetanus antitoxin, 40,000 units were given intravenously and 40,000 units intramuscularly. Intravenous fluids were given, and the patient resumed taking of 300,000 units of penicillin daily. Mephenesin (tolserol®), a myonesin product, was given for 24 hr., initially 2 gm. intravenously and after five minutes, when the trismus disappeared, 3 gm. orally. The medication was repeated intravenously at intervals of one to three hours in order to relieve the trismus.

The next day, when generalized spasms appeared, sedation therapy was instituted, with tribromoethanol, 30 mg. per kilogram, and pentobarbital rectally, 3 grains (0.2 gm.) every four hours. With the onset of generalized spasms, periods of dyspnea developed, and the pharynx and laryngotracheobronchial tree were full of mucus. Postural drainage and nasal

pharyngeal aspiration with a rubber catheter failed to provide relief, and tracheotomy was done 20 hr. after the onset of the generalized spasms. It was found necessary to clear the tracheobronchial tree frequently of secretion by means of suction.

For the next six days, generalized spasms continued to occur about every two hours in spite of heavy sedation with tribromoethanol and pentobarbital. On the seventh day, the tribromoethanol was discontinued and shortly thereafter oral feeding was begun, although the patient was kept under sedation with pentobarbital, 4.5 grains (0.3 gm.) three times a day. Spasm reappeared only occasionally and disappeared completely on the 18th day. Two days later, decannulation was done.

Fifty-three days after the onset of the tetanus, trismus was still present but the patient was up and around and the toes were almost healed.

CASE 3.—G. M., a Negro boy aged 6 yr., was admitted to the hospital Aug. 17, 1949, at 11 a. m., because of trismus, stiff neck, and rigid back, which appeared that morning. He was alert. The mother said that four days previously he received a cut behind the right ear when struck with a stone. This was treated elsewhere, and no tetanus antitoxin was given. The wound became infected and was redressed on August 16.

On admission, the temperature was 98.6 F., the pulse rate 100, and the respiration rate 28. There was a 2 cm. laceration behind the right ear, with purulent discharge. The neck and back were rigid. Severe trismus prevented examination of the mouth and pharynx. The urine was normal. The blood count revealed 4,100,000 red blood cells, 13,800 leukocytes with 78% polymorphonuclear neutrophils, 6% eosinophils, 13% lymphocytes, and 1% monocytes. Serologic examination showed a quantitative Kahn reaction of 4 units with a negative Wassermann reaction. He had within the past year completed treatment for congenital syphilis. The spinal fluid showed a negative reaction to the Wassermann test, normal protein and dextrose, no cells, and a negative Pandy test reaction.

The patient was transferred to a dark, quiet room, and immediate treatment was instituted consisting of 40,000 units of tetanus antitoxin intramuscularly and 40,000 units intravenously in 200 cc. of isotonic sodium chloride solution with 1 cc. of 1 : 1,000 epinephrine. Secobarbital (seconal®), 3 grains (0.2 gm.), as a suppository was followed by tribromoethanol, 0.770 gm. given by way of a rectal tube. Aqueous penicillin in doses of 100,000 units and crysticillin® penicillin G procaine for aqueous injection, 300,000 units, were given intramuscularly.

Two hours later, the first generalized tonic seizure with flexed arms, arched back, and abdominal rigidity occurred and lasted about two minutes. The patient complained of abdominal pain and became very restless. The pain was controlled with 0.770 gm. of tribromoethanol rectally. Four hours later, severe spasms with risus sardonicus and opisthotonos recurred. Salivation was profuse, and a large amount of mucus was aspirated with a nasal catheter. That evening, sodium amobarbital (sodium amytal®), 2 grains (0.12 gm.), was given intravenously to relieve the frequent tonic crises. At midnight, the spasms were accompanied by periods of respiratory arrest and cyanosis lasting as long as one minute. Laryngospasm with respiratory stridor appeared. Sodium amobarbital, 4 grains (0.25 gm.), were given intravenously. At this time the consultant in otolaryngology performed a tracheotomy and aspirated several cubic centimeters of thick tenacious secretion from the trachea. Postural drainage was then instituted. The breathing immediately became easier and spasms were infrequent and mild. For 30 hr. after the tracheotomy no sedation was required. The patient remained greatly depressed, and the spasms were mild.

On August 20, the tongue slipped between the teeth and was bitten almost in half. The next day a total of 2.25 cc., or 6.75 mg., of tubocurarine (*d*-tubocurarine®) induced enough relaxation to permit replacing the tongue, but shortly after respirations ceased, necessitating the use of oxygen and artificial respiration for a period of 15 min.

During the following 10 days, the spasms were controlled with pentobarbital, 3 grains (0.2 gm.), rectally four times a day, and tribromoethanol, 0.770 gm. twice a day. The patient remained comatose. On the 21st hospital day he was awake and alert. Intravenous protein and vitamin therapy was adequate, as shown by the blood-chemistry readings: total protein, 5.8 mg. per 100 cc. chlorides, 98.2 mg., and calcium, 9.1 mg.

During the next eight days, the spasms were controlled with pentobarbital, 3 grains (0.2 gm.) every three hours. Tube feedings were given. Forty-one days after admission, the patient

was able to swallow and speak and was free of spasms without sedation. On the 51st day after the tongue was repaired, the tracheotomy tube was removed. The patient was discharged, after extensive physical therapy care for drop foot, on November 19, exactly 94 days after admission.

CASE 4.—L. W., a Negro woman aged 65, was admitted to the hospital on Jan. 25, 1950, at 9 a. m., complaining of pain and tenderness of the left lower molar teeth for two weeks. She stated that five days previously she awoke with her mouth twisted over to the right and that she was unable to swallow even liquids. On admission, she was having difficulty opening her mouth.

Examination revealed a mentally alert patient with a temperature of 100.6 F., pulse rate of 100, respiration rate of 20, and blood pressure of 110/70. There were trismus and tenderness of the left lower jaw. A slight swelling was present over the left lower mandibular region, and the head was held in extension. The left pupil was 3 mm. in diameter and failed to react to light. The right pupil reacted normally. The other reflexes were normal. The urine showed albumin (1 plus). The Kahn reaction was negative.

Five per cent dextrose and saline fluids were given intravenously immediately, and 300,000 units of penicillin were given intramuscularly. Twelve hours after admission, generalized spasms appeared and a diagnosis of cephalic tetanus was made. The patient was then given tetanus antitoxin, 40,000 units intravenously and 40,000 units intramuscularly, tribromoethanol, 30 mg. per kilogram rectally, and pentobarbital, 3 grains (0.2 gm.) every four hours, and amobarbital 7.5 grains (0.5 gm.) intravenously as needed.

One and one-half hours later, typical laryngospasm, accumulation of secretion in the pharynx, and periods of cyanosis developed. Twenty-five degree postural drainage and nasopharyngeal suction with a rubber catheter were instituted. The next day breathing became irregular and then ceased for several seconds but was promptly restored with 0.5 cc. of nikethamide (coramine®) intravenously. Sedation was discontinued because the patient was comatose with absent corneal reflexes and fixed eyeballs.

Tracheotomy was performed with the consent of the attending physician 20 hr. after the first generalized spasm appeared. At this time, owing to the abundance and tenacity of the secretion, it was necessary to perform bronchoscopy through the tracheal stoma. Thereafter, aspiration with the rubber catheter sufficed. The patient remained comatose for three days, during which time no sedation was given. The tracheobronchial tree was frequently aspirated. The trismus persisted, but no generalized spasms occurred after the tracheotomy, even without sedation.

During the afternoon of the fourth hospital day, the patient began to respond to questions. On the sixth day, Levine tube feedings were started because of the persistent trismus. By the 19th day, the mouth could be opened voluntarily for a distance of 1 in. (2.5 cm.) and decannulation was done. Six days later, because of extensive cavities of the lower teeth and roentgenological evidence of periapical abscesses of the left lower molars, all these teeth were extracted.

The patient was discharged on the 34th hospital day, completely well and with no trismus or other residual effects.

CASE 5.—M. J., an unmarried Negro girl aged 16, was admitted to the hospital on June 6, 1950, with complaints of sore throat and low back and abdominal pain for two days. She had been in the hospital two months previously with an incomplete abortion of a 4½-mo.-old fetus. No information as to instrumentation or any other precipitating cause of her present complaints could be elicited. In the hospital the abortion spontaneously became complete, and recovery was uneventful.

Upon examination the temperature was found to be 99.2 F., the pulse rate 104, the respiration rate 22, and the blood pressure, 102/70. There was marked rigidity of the back muscles and hypogastric tenderness. On pelvic examination a tender but movable mass, 5 by 5 cm., was palpable in the right adnexa. There were no noteworthy laboratory findings. The diagnosis was inflammatory pelvic disease.

The following day she developed marked nuchal rigidity. The results of spinal-fluid examination were normal. On the third hospital day she had trismus and generalized spasms with opisthotonos. Hyperactive deep reflexes and bilateral ankle clonus were noted at this time. Mucus accumulated in the pharynx followed by cyanosis and respiratory arrest lasting

several seconds. At this time a diagnosis of severe tetanus was made and immediate treatment was instituted, consisting of tetanus antitoxin, 40,000 units intramuscularly and 40,000 units intravenously, penicillin in full dosage, postural drainage, oxygen, and sedation with pentobarbital, 3 grains (0.2 gm.) every four hours, and tribromoethanol, 1.5 gm. rectally. Because of the continued cyanosis and accumulation of mucus, tracheotomy was performed. Pentobarbital in 1.5 grain (0.1 gm.) doses was given every four hours. Several generalized tetanic spasms occurred daily for the next five days. Thereafter, the spasms were localized but trismus and abdominal rigidity continued to the 14th hospital day. On the 15th day she was able to take nourishment by mouth and the tracheotomy cannula was removed. On the 23d hospital day she was discharged, apparently well.

CASE 6.—L. B., a Negro woman aged 50, was admitted to the hospital on Aug. 19, 1950, at 4 p. m., with complaints of backache, inability to walk, and "lockjaw" for two days. The only history of injury was a knife cut on the left thumb one month before, which had healed. She was well up to one week before admission, when she noticed weakness in the lower extremities and stiffness in the neck, and she feared going to sleep because she might bite her tongue.

The initial examination revealed a conscious, oriented patient with marked opisthotonos, neck rigidity, boardlike hardness of the abdomen, and trismus. The rectal temperature was 100 F., the pulse rate 120, and the respiration rate 26 per minute. Several localized spasms occurred during the examination. She was given secobarbital, 4.5 grains (0.3 gm.) rectally,

TABLE 1.—*Historical Review*

Thomas Curling	1837	Suggested tracheotomy
G. M. Humphry	1856	Unknown number of patients
C. C. Bustillo	1943	1 patient survived
Harris, Montreuil, and McDermott	1948	2 patients survived
Turner and Galloway	1949	1 patient survived
Creech, Woodhall, and Ochsner	1950	5 patients survived
Tucker and Lasater	1950	2 patients survived

tetanus antitoxin, 40,000 units intravenously and 40,000 units intramuscularly, and penicillin in full dosage. The foot of the bed was elevated.

Generalized spasms appeared five hours after admission, during which the tongue was injured. Secretions welled from the mouth, and during the seizures breathing ceased and cyanosis was evident. Sodium thiopental (sodium pentothal®) 7.5 grains (0.5 gm.), was given intravenously, and tracheotomy was performed. Tracheobronchial aspiration removed much accumulated mucus. Humidified oxygen administered by way of the tracheal cannula was started.

During the next 10 days her temperature remained around 102 F. Incontinence of urine and feces persisted for four days. During this time pentobarbital, 1.5 grains (0.1 gm.), was given four times a day and only localized spasms were seen. On the fifth day the use of intravenous fluids was supplemented by small amounts given by mouth, and thereafter Levine-tube feedings were given until the ninth day when a liquid diet could be taken orally. On the 16th hospital day the patient was able to swallow readily and was able to breathe with the cannula closed; so decannulation was done. The patient was discharged, completely well, on the 25th hospital day.

HISTORICAL RÉSUMÉ

Thomas Curling,[5] in his treatise of 1837, suggested the use of tracheotomy in tetanus in these words:

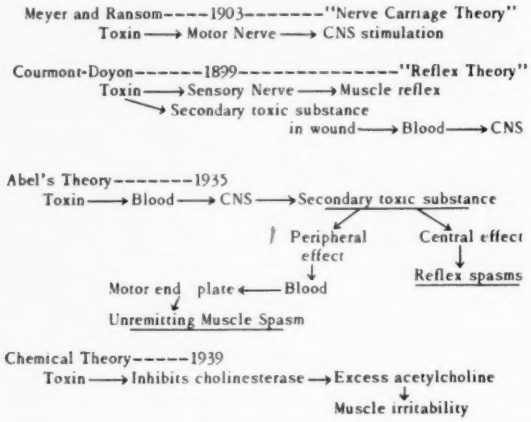
There is a sense of constriction at the throat, an apprehension of suffocation, and when the paroxysm ensues, all the outward signs of asphyxia are present. I have watched the tetanic patient in the sufferings of this dreadful moment. I have seen him tear away the coverings,

5. Curling, T. B.: A Treatise on Tetanus, Philadelphia, Haswell, Barrington & Haswell, 1837.

grasp his throat with suffused and livid countenance, and in all the agonies of impeded respiration, struggle for breath and for life. In a case of imminent danger from suffocation, it has been suggested that relief might be afforded by an opening made in the trachea.

In 1856, Humphry [6] reported tracheotomy in the total treatment of tetanus. In 1943, Bustillo [7] performed a tracheotomy on a patient with tetanus with glottic spasm. In 1948, Harris, McDermott, and Montreuil [8] reported two cases of survival. Turner and Galloway [9] reported a patient with tetanus which required tracheotomy and the use of a Drinker respirator. Recently, Creech, Woodhall, and Ochsner [10] reported six cases of tracheotomy with one death. Tucker and Lasater [11] performed tracheotomies on three patients with tetanus with one fatality.

TABLE 2.—*Theories of Pathogenesis*


Meyer and Ransom----1903-------"Nerve Carriage Theory"
Toxin → Motor Nerve → CNS stimulation
Courmont-Doyon------1899----------"Reflex Theory"
Toxin → Sensory Nerve → Muscle reflex
Secondary toxic substance
in wound → Blood → CNS
Abel's Theory-------1935
Toxin → Blood → CNS → Secondary toxic substance
Peripheral effect
Central effect
Reflex spasms
Motor end plate ← Blood
Unremitting Muscle Spasm
Chemical Theory-----1939
Toxin → Inhibits cholinesterase → Excess acetylcholine
Muscle irritability

PATHOGENESIS

The pathogenesis of tetanus is still in doubt and is a subject of much controversy. The currently accepted theories are given in brief. According to the nerve carriage theory of Meyer and Ransom,[12] the tetanus toxin reaches the central

6. Humphry, G. M.: Cases of Tracheotomy in Laryngitis for Foreign Body, and in Tetanus, London M. A. J. **4**:158-160, 1856.

7. Bustillo, C. C.: Tetanos, espasmos de la glotis, traqueotemia, curación: caso clinico, Revista méd.-quir. Oriente **4**:233-238 (Dec.) 1943.

8. Harris, R. C.; McDermott, T. F., and Montreuil, F. L.: The Treatment of Tetanus, Pediatrics **2**:175-185 (Aug.) 1948.

9. Turner, V. C., and Galloway, T. C.: Tetanus Treated as a Respiratory Problem, Arch. Surg. **58**:478-483 (April) 1949.

10. Creech, O.; Woodhall, J. P., and Ochsner, A.: The Necessity for Tracheotomy in the Treatment of Tetanus to Prevent Lethal Respiratory Complications, Surgery **27**:62-73 (Jan.) 1950.

11. Tucker, J. S., and Lasater, G. M.: The Treatment of Tetanus, Journal-Lancet **70**:107-110 (March) 1950.

12. Meyer, H., and Ransom, F.: Untersuchungen über den Tetanus, Arch. exper. Path. u. Pharmakol. **49**:369-416, 1903.

nervous system by way of the peripheral motor nerves and spreads along the neural pathways to the central nervous system. According to the reflex theory of Courmont and Doyon,[13] the unneutralized tetanus toxin stimulates the sensory-nerve endings in muscle, producing localized tetanus, and because of a secondary toxic substance which is transmitted by way of the circulation, generalized tetanus occurs. Abel and Hampil's theory [14] is that the unchanged tetanus toxin formed at the site of the original lesion is distributed by means of the circulation to the central nervous system. The so-called secondary toxic substance is not formed in muscle but in the central nervous system. The following conclusions are drawn by Abel and associates [15]:

> The toxin exhibits both central and peripheral action. Each action may be demonstrated independently of the other. The central effect characterized by reflex motor convulsions is due to poisoning of the cells of the spinal cord, medulla and pons. The peripheral effect recognized as unremitting rigidity of voluntary muscles results from fixation of the toxin upon the motor end organs.

The theories of other investigators [16] propose that tetanus toxin destroys cholinesterase, thereby increasing acetylcholine in the muscles and in the central nervous system.

It is probable that the true nature of the pathogenesis of tetanus is a composite of the above theories, and recent authors [17] support one or another of them.

PATHOLOGY

Since the effect of tetanus on the central nervous system is toxic and not inflammatory, there are no severe morphologic changes.[18] According to Baker,[4] if detailed pathological studies are made of patients in whom the illness has lasted at least three days, definite changes within the nervous system are evident. On gross examination of 12 patients he found petechiae throughout the brain, and in four cases there were signs of definite hemorrhage. Microscopically, even as early as the third day of illness, nerve cells, especially those near blood vessels, showed swelling, chromatolysis, and detached cell processes. These changes occurred predominantly in the dorsal motor nucleus of the 10th cranial nerve and in the

13. Courmont, J., and Doyon, W.: Le tetanos: Étiologie, pathogénic, pronostic, traitement, Paris, J.-B. Ballière & fils. 1899.

14. Abel, J. J., and Hampil, B.: Researches on Tetanus, Bull. Johns Hopkins Hosp. **57**:343-372 (Dec.) 1935.

15. Abel, J. J.; Firor, W. M., and Chalion, N.: Researches on Tetanus, Bull. Johns Hopkins Hosp. **63**:373-403 (Dec.) 1938.

16. Harvey, A. M.: Peripheral Action of Tetanus Toxin, J. Physiol. **96**:348-365 (Aug. 14) 1939. Bayne-Jones, S.: Tetanus (Ludvig Hektoen Lecture), Proc. Inst. Med. Chicago **14**:98-107 (April 15) 1942.

17. Göpfert, H., and Schaefer, H.: Über die Mechanik des Wundstarrkrampfes, Arch. Exper. Path. u. Pharmakol. **197**:93-122 (Dec.) 1940. Schmidt, S.: Zur Fragen der Bindung in Toxin on Hämoglobin beim Tetanus, Ztschr. Immunitätsforsch. **103**:236-240 (July) 1943. Roofe, P. S.: The Affinity of the Nerve Fiber for Tetanus Toxin, J. Kansas M. Soc. **49**:60-61 (Feb.) 1948.

18. Grinker, R. R.: Neurology, ed. 3, Springfield, Ill., Charles C Thomas, Publisher, 1945. Lichtenstein, B. W.: A Textbook of Neuropathology, with Clinical, Anatomical and Technical Supplements, Philadelphia, W. B. Saunders Company, 1949, pp. 168-169. Minassian, P.: Über die histologischen Veränderungen des nerven Systems beim Tetanus, Zentralbl. allg. Path. **15**:692, 1904.

motor nucleus of the fifth nerve, although changes were also present in the anterior horn cells. He noted many areas of demyelination with no cellular reaction within the midbrain, pons, and medulla. After the eighth day, numerous glial nodules were found within the medulla, occasionally involving the cranial nuclei. From his studies Baker [19] concludes that most of the cellular alterations, consisting of swelling and perivascular chromatolysis, are reversible. Moreover, the anterior horn cells are much less affected than the cells of the cerebral cortex and the cells of the cranial-nerve nuclei.

TABLE 3.—*Pathologic Physiology*

	Inspiration	Expiration
I. Peripheral neuromuscular dysfunction		
A. Muscles of quiet respiration		
	Diaphragm	Relaxation of inspiratory muscles
	External-intercostals	Internal intercostals
B. Accessory muscles of forced respiration		
	Sternocleidomastoid	Rectus abdominis
	Scaleni	External oblique
	Pectoralis minor	Internal oblique
	Levator costarum	Transverse abdominis
	Serratus posterior superior	
II. Central neural dysfunction in brain stem		
A. Respiratory center		
B. Swallowing center		
Pharyngeal reflex		
Esophageal reflex		
C. Trigeminal nerve motor nucleus		
III. Mechanical obstruction		
A. Vocal-cord spasm		
B. Tracheobronchial secretions		
C. Tongue obstruction		
D. Trismus factor		
IV. Chemical physiology alteration		
A. Hypercapnia		
B. Anoxemia		
C. Acidemia		
V. Secondary pulmonary complications		
A. Atelectasis		
B. Pulmonary edema		
C. Bronchopneumonia		

PATHOLOGIC PHYSIOLOGY

Relevant to the pathologic physiology of tetanus is Humphry's [6] description of a patient in 1856, almost a century ago.

> The spasms affected the neck and body to a certain extent, but were much more severe in the lips, fauces and larynx. When they were at their height, she could not breathe, owing to the firm closure of these parts. She struggled hard and turned blue in the face. They came on whenever she dropped asleep or lay down; also whenever she attempted to expectorate the mucus collected in the air passages, and gave great trouble. As she was decidedly getting worse, in spite of the various means adopted to relieve her, I determined to try the effect of opening the trachea, which would at any rate remove the apprehension of choking and might afford a more easy outlet for the accumulating mucus.

19. Baker, A. B.: Medullary Involvement in Tetanus, Am. J. Path. **19**:709-724 (July) 1943.

In discussing the pathologic physiology of tetanus our attention is focused on the important role of the respiratory system. Several authors [20] recently have discussed respiratory obstruction in bulbar poliomyelitis, in which the factors of obstruction by secretion and those of asphyxia are identical with those seen in tetanus. With the onset of generalized, or convulsive, tetanus, which may follow the early, or localized, type, the problem of difficulty of respiration becomes of major concern and will be discussed under the headings of (1) peripheral neuromuscular dysfunction; (2) central or brain-stem dysfunction; (3) mechanical obstruction; (4) chemical physiological alteration; (5) secondary pulmonary complications.

1. *Peripheral Neuromuscular Dysfunction.*—The spasms in tetanus have a profound effect on the muscles of inspiratory and expiratory excursion. Numerous physiologists [21] discuss the mechanism of normal respiration. According to Fulton, in quiet inspiration the diaphragm descends and the external intercostal muscles contract to elevate the ribs anteriorly, thereby enlarging the chest cavity. In the infant, because of the horizontal plane of the ribs, respiration is abdominal or diaphragmatic. In forced inspiration, the first rib and sternum are elevated by the sternocleidomastoid muscles and the scaleni and pectoralis-minor muscles. The other ribs are moved toward the first rib by the external intercostal muscles. The levatores-costarum and serratus-posterior superior muscles extend downward from the vertebral column to the ribs and thus also aid inspiration. Quiet expiration is mainly passive and is brought about by relaxation of the inspiratory muscles, but there is some contraction of the expiratory internal intercostal muscles. In forced expiration, the main muscles are the internal and external obliquus, the rectus abdominis, and the transversus abdominis muscles of the abdominal wall.

In addition, according to Best and Taylor, in normal muscle movements reflex inhibition of the antagonists accompanies reflex excitation of the protagonists. For example, the abdominal muscles, which are the antagonists of the diaphragm, relax as the diaphragm descends in inspiration; however, in tetanus the normal respiratory physiology is very seriously disturbed. There is tonic contraction of the muscles with, usually, simultaneous contraction of the protagonists and antagonists. Thus it can be readily appreciated that in clinical tetanus nuchal rigidity and opisthotonos limit expiration and abdominal rigidity interferes with inspiration and so contribute to asphyxia. Turner and Galloway [9] report an instance in which respiration ceased because of spasms of the chest and abdominal musculature.

2. *Central or Brain-Stem Dysfunction.*—It has been shown by histopathological study [4] that the dorsal motor nucleus of the vagus nerve is frequently selectively damaged by tetanus toxin. There is also demyelination of neuron pathways in

20. (*a*) Galloway, T. C., and Seifert, M. H.: Bulbar Poliomyelitis, J. A. M. A. **141**:1-8 (Sept. 3) 1949. (*b*) Priest, R. E.; Boies, L. R., and Goltz, N. F.: Tracheotomy in Bulbar Poliomyelitis, Ann. Otol. Rhin. & Laryng. **56**:250-263 (June) 1947. (*c*) Fox, M. J., and Sanders, D. D.: The Management of Respiratory Embarrassment in Poliomyelitis, Wisconsin M. J. **46**:885-894 (Sept.) 1947.

21. (*a*) Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, ed. 5, Baltimore, Williams & Wilkins Company, 1950, pp. 344-441; (*b*) ibid., p. 494. (*c*) Wright, S.: Applied Physiology, ed. 8, New York, Oxford University Press, 1945, pp. 605-640. (*d*) Wiggers, C. J.: Physiology in Health and Disease, ed. 5, Philadelphia, Lea & Febiger, 1949, pp. 414-419. (*e*) Nims, L. F., in Howells, W. H.: A Textbook of Physiology, edited by J. F. Fulton, ed. 16, Philadelphia, W. B. Saunders Company, 1949, pp. 782-797.

the midbrain, pons, medulla, and cord. This accounts for the occurrence of pharyngeal, laryngeal, and esophageal paralysis in tetanus. With severe pharyngeal paralysis, nasal and pharyngeal secretions collect in the pharynx and overflow into the larynx and tracheobronchial tree. The normal salivary secretion alone amounts to 1,000 to 1,500 cc. daily.[21b] To this is added the secretion of the numerous mucous glands in the nose, mouth, nasopharynx, and oropharynx. The adductors of the vocal cords are normally stronger than the abductors, so that if both are weakened by tetanus toxin acting on the laryngeal nerves and then spasm occurs, adduction results and obstructs breathing.[22] In addition, the mere presence of secretion causes stimulation by way of the superior laryngeal nerve so that reflexes of choking, gagging, and coughing occur with closure of the glottis. This may be seen, for example, in everyday experience when liquids go down the "wrong way."

Brain-stem damage includes damage of the respiratory, the cough reflex, and the swallowing centers. Respiration may become irregular in rate, depth, and rhythm. Periods of apnea then intervene. In such cases, it is lifesaving to institute artificial respiration by means of positive pressure through the tracheotomy cannula[8] or by the use of the Emerson respirator. With central diminution of the cough reflex, secretion accumulates in the tracheobronchial tree. The significance of this can be appreciated readily in the causation of atelectasis or aspiration pneumonia. As for swallowing dysfunction, there is not only a loss of normal secretion disposal with consequent pooling in the hypopharynx but also the problem of nutrition which must be solved by parenteral administration of fluid or by tube feedings.

3. *Mechanical Obstruction of the Airway.*—This occurs from vocal-cord spasm and obstruction of the tracheobronchial tree by secretion. Also, the tongue may be frequently injured during the convulsive seizures and thus cause obstruction of the airway by blood, as seen by Hartmann[23] and also noted by us (Case 3). Adequate sedation for the suppression of tonic crises often causes such relaxation of the tongue that the attached epiglottis covers the glottic airway. Trismus prevents the introduction of the commonly used oropharyngeal airway. As a consequence of respiratory obstruction, there is decreased availability of oxygen, especially at a time when the nervous tissue has already been damaged by the toxin. Of all body tissues, neural tissue is the least capable of withstanding the lack of oxygen.

4. *Chemical Physiological Alteration.*—In a patient gravely ill with tetanus it is difficult to identify the early signs of anoxia, such as headache, restlessness, irrationality, disorientation, and dyspnea.[21a] At this stage the elevation of blood pressure and the use of a photoelectric oximeter may yield valuable indications. The late signs of anoxia are lethargy, cyanosis, coma, and shock.[21a]

Gray,[24] in a recent work on the physiology of respiratory obstruction, shows that asphyxia has three components: hypercapnia, anoxemia, and acidemia. Hypo-

22. Nelson-Jones, A., and Williams, R. H. H.: Tracheotomy in Bulbar Poliomyelitis, Lancet **1**:561-563 (May) 1945.

23. Hartmann, A. F.: Conference at the St. Louis Children's Hospital, June 30, 1948: Case 4, J. Pediat. **33**:646-561, 1948.

24. Gray, J. S.: The Physiology of Respiratory Obstruction, Ann. Otol. Rhin. & Laryng. **59**:72-77 (March) 1950.

ventilation causes retention of carbon dioxide in the body tissues which results in an increase in the carbon dioxide of the blood. This elevation of arterial carbon dioxide is known as hypercapnia. Anoxemia is a fall in the arterial oxygen saturation from hypoventilation. Hypercapnia and anoxemia produce acidemia, which is a fall in the pH of the blood. Acidemia in tetanus is caused by (1) diminished carbon-dioxide elimination, (2) diminished oxygen availability, and (3) accumulation of excess lactic acid from tetanic muscle spasm. Gray distinguishes between the early signs of hypercapnia and anoxemia. Hypercapnia causes restlessness, disorientation, apprehension, and later respiratory depression and circulatory collapse. On the other hand, anoxemia produces a gradual lapse into unconsciousness without apparent distress, as seen in high-altitude flying without oxygen supplement.

5. *Secondary Pulmonary Complications.*—These inevitably appear if the previously mentioned pathophysiological changes are allowed to persist. Laryngeal and tracheal obstruction produces anoxic anoxia or inadequate oxygenation of the blood in the lungs.[21a] The resultant anoxemia damages the respiratory center, which in turn induces more anoxemia. Anoxemia also increases the permeability of the pulmonary capillaries, and this encourages edema of the lungs, which further impairs gaseous exchange and induces more anoxemia. When there is stagnation of secretions in the bronchial tree, bronchopulmonary segments become isolated from their air supply, oxygen is absorbed, and atelectasis results. Massive atelectasis may be the direct cause of death, as seen in Case 1.

It has been shown by Galloway and Seifert [20a] that postural drainage combined with aspiration of secretion from the tracheobronchial tree can be effective only if the foot of the bed is elevated so that the patient lies at an angle of 30 to 35 degrees with the horizontal. This large angle is necessary to overcome the normal 20 degree angle of elevation between the tracheobronchial tree and the horizontal body axis. In a recent discussion on postural drainage, Galloway [25] has revised his figure to 25 degrees, which we have found to be adequate and more easily maintained. Aspiration with the nasal catheter merely clears the nasopharynx, and contact with the vocal cords may precipitate fatal laryngospasm. Moreover, nasopharyngeal aspiration cannot effectively remove secretion from the tracheobronchial tree, and therefore in the presence of these secretions only aspiration through the tracheal cannula can help to restore and maintain normal respiratory function. Finally, patients allowed to aspirate pharyngeal secretion and to retain tracheobronchial secretion tend to have bronchial or aspiration pneumonia.

TREATMENT

The indications for tracheotomy in tetanus depend upon neuromuscular dysfunction and mechanical obstruction. They are (1) prolonged spasm of the muscles of respiration, (2) absent cough reflex, (3) absent swallowing reflex, (4) laryngeal obstruction, (5) secretion in the tracheobronchial tree, (6) tongue trauma, and (7) coma.

In tetanus we feel that tracheotomy should be done before irreversible respiratory decompensation occurs owing to any or all of the above factors. Trismus, in addition, precludes the advantage of tracheotomy over the bronchoscope. How-

25. Galloway, T. C., in Simpson, J. R.: Management of Acute Laryngotracheo-Bronchitis, Arch. Otolaryng. **50**:724-731 (Dec.) 1949.

ever, it is often necessary later in order to remove secretions to bronchoscope the patient through the tracheal stoma. In this respect, we cannot agree that routine tracheotomy should be done merely because of the presence of trismus and dysphagia.[10] It is well known that some cases of tetanus are mild and do not approach respiratory decompensation.

Surgical excision of the wound in an attempt to eradicate the source of toxin production is still carried out by numerous authors.[26] It has been shown [27] by animal experimentation that amputation in no way changed the progress of the disease compared with no amputation in the controls. This is confirmed in the case of human beings and animals by Abel and Hampil.[14] Gessler [28] and Spaeth [29] show that administration of tetanus antitoxin neutralizes any toxin which may be discharged from the wound site. Only the adherence to good surgical principles should guide one in the care of the local wound, and deforming excisions are unwarranted. Thus, removal of foreign bodies and drainage of abscesses, if present, must be done.

Furthermore, let us consider the effect of current tetanus treatment on respiratory function. Curare is used to block the transmission of nerve impulses at the

TABLE 4.—*Treatment*

1. Tracheotomy at time of respiratory decompensation
2. Sound surgical principles in care of local wound
3. Tetanus antitoxin
4. Optional use of curarization
5. Sedation with barbiturates cautiously
6. After care of tracheotomized patient
   a. Aspiration as necessary
   b. Postural drainage
   c. Securing patient to bed
7. Nutrition and parenteral administration of fluid

myoneural junction by counteracting acetylcholine. The chief danger in the use of this drug is the narrow margin of safety between the desired muscular relaxation and paralysis of the muscles of respiration.[30] If curare is used, neostigmine should be at the bedside and one should be prepared to administer artificial respiration.

Sedation with tribromoethanol and the barbiturates, which is necessary for the control of increased nerve irritability and tetanic convulsive seizures, impairs respiratory function. The optimum dose is one that controls spasms and seizures without dangerously depressing the rate and depth of respiration. One must avoid apnea resulting from the effect of excessive sedation on the respiratory center. Moreover, barbiturate action combined with stimulation of the vocal cords by the pharyngeal catheter may produce a fatal laryngospasm. This laryngospasm may be averted by tracheotomy. Deep sedation prevents the patient from coughing

26. Creech, Woodhall, and Ochsner.[10] Vinnard, R. T.: Three Hundred Fifty-Two Cases of Tetanus, Surgery **18**:486-492 (Oct.) 1945. Tucker and Lasater.[11]

27. McClintock, C. T., and Hutchings, W. H.: The Treatment of Tetanus, J. Infect. Dis. **13**:309-320, 1913.

28. Gessler, C. N.: A Clinical Study of Tetanus: An Analysis of 121 Cases of Tetanus, J. Tennessee M. A. **38**:363-365 (Nov.) 1945.

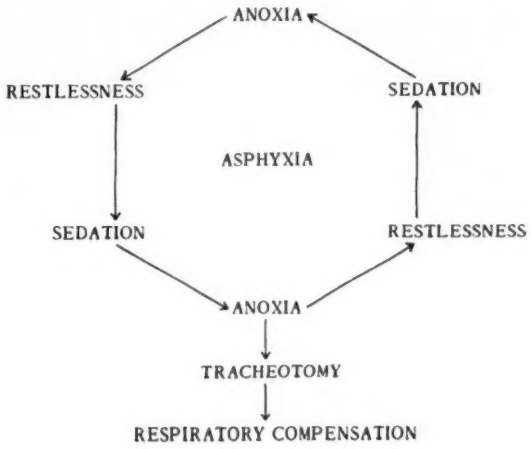
29. Spaeth, R.: A Clinical Study of Tetanus, Am. J. Dis. Child. **60**:130-169 (July) 1940.

30. Ory, E. M., and Grossman, L. A.: The Management of Tetanus with Curare: Report of 2 Cases, Am. J. M. Sc. **215**:448-450 (April) 1948.

and emptying his bronchioles and also prevents the use of the accessory muscles of respiration. This further emphasizes the need for tracheal stoma aspiration. Animal experiments by Firor, Lamont, and Shumaker [8] support these views:

> Although the utmost care must be taken in applying to human beings the results of experiments on dogs, our observations on the manner of death and on the effect of respiratory depressants suggest that such drugs should be used with extreme caution in cases of human tetanus.

It is seen, therefore, that a vicious circle is established whereby barbiturates further depress respiration, increasing the hypercapnia and anoxemia with restlessness, thereby requiring more barbiturate and producing more asphyxia until death ensues. Tracheotomy can interrupt this circle by alleviating this asphyxia (chart). Others [8] have shown that the optimum dosage was reduced to about one-third of that used before tracheotomy, and we have found that sedation can be more safely administered and in an appreciably smaller amount after tracheotomy.



Vicious cycle to be avoided.

AFTERCARE

The after care of the tracheotomized patient with tetanus deserves comment. Our routine is to elevate the foot of the bed to 25 degrees from the horizontal. Since measuring the angle of elevation with a giant protractor is impractical, we utilize the fact that the value of the trigonometric function of the sine angle 25 degrees is equal to 0.4226. This factor, when multiplied by the length of the bed, accurately gives the desired elevation. For example, a 6-ft. bed should be elevated 2.4 ft. We secure the patient's feet to the foot of the bed with a roller bandage in the form of a Collin's hitch, making sure first to wrap each ankle with a towel. An excellent diagram of Collin's hitch may be found in Magnuson's book.[31] Nutrition is maintained by adequate fluid administered parenterally during the period of generalized

31. Magnuson, P. B.: Fractures, ed. 4, Philadelphia, J. B. Lippincott Company, 1942, p. 235.

tonic spasms. This may be supplemented or replaced by Levine-tube feedings. It is important to lower the bed to the horizontal for an hour after each feeding to prevent gravitational regurgitation of the feeding. With the patient in the postural-drainage position, aspiration through the cannula should be done whenever secretions are present. If this is inadequate, bronchoscopic aspiration through the stoma should be done. Moreover, the secretions should be kept thin by a humid atmosphere and, if necessary, by irrigation with 4 cc. of 3% warmed sodium bicarbonate solution followed by 4 cc. of isotonic sodium chloride solution. Decannulation should not be carried out until the patient can swallow and speak and has had no spasms for 24 hr. without sedation.

SUMMARY

Six patients with tetanus who required tracheotomy for survival are discussed.

The historical background of tracheotomy in tetanus is reviewed.

The theories of pathogenesis and the pathology of tetanus are discussed.

The pathologic physiology as a respiratory problem and its relation to treatment are outlined.

The value of postural drainage, tracheotomy, and aspiration are discussed and the danger of oversedation is emphasized.

1835 W. Harrison St.

55 E. Washington St.

25 E. Washington St.

TREATMENT OF HAY FEVER BY HYPOSENSITIZATION

SOLOMON SLEPIAN, M.D.
BROOKLYN

OF THE various allergic conditions that allergists are called upon to treat, the best results are obtained in the management of seasonal hay fever.

In treating patients with seasonal hay fever by injections, the allergist does not completely desensitize. He simply makes the patient less sensitive to the noxious pollens to which he may be hypersensitive.

The mechanism by which this is accomplished depends upon the formation in the body of a blocking thermostabile antibody. It is produced by the repeated injections of pollen antigen. This blocking antibody should not be confused with the skin-sensitizing antibody, which is responsible for the production of symptoms when skin-sensitizing antibody combines with antigen and liberates histamine or "H"-substance. There are indications that another still unknown immunologic mechanism exists which is responsible for protection. This must be so, for there are patients who have no demonstrable blocking antibody and who, nevertheless, obtain relief from specific treatment.

While pollens are the commonest causes of seasonal hay fever, there are other possible excitants; molds, insects, and foods may play a part. The pollens of importance are those which are air-borne. As a rule, the pollens produced by fragrant and colorful flowers and shrubs are small in amount and usually sticky. They are disseminated by insects, thus rarely coming in contact with human beings who might be sensitive to this type of pollen. The inconspicuous weeds and grasses must depend on wind for distribution, and they produce tremendous quantities of pollen, thus creating a health hazard.

There are three pollen seasons, designated as spring, early summer, and late summer.

For the Middle Atlantic states, spring hay fever, occurring from early April to early June, is usually caused by tree pollen. The grass pollens, including plantain, cause symptoms in the late spring and in the summer, the season extending from the middle of May to the end of June. The weed pollens are responsible for symptoms in the late summer and fall, from mid-August to early October.

For New York and its vicinity, the most important tree pollens are oak, hickory, birch, ash, and beech, while elm, poplar, maple, walnut, and elder are of secondary importance. The important grass pollens are sweet vernal, junegrass, orchard grass, redtop, and timothy.

Dr. Slepian is Chief, Allergy Clinic, Brooklyn Eye and Ear Hospital.

Read before the Clinical Conference, Department of Otolaryngology, Brooklyn Eye and Ear Hospital, Brooklyn, April 11, 1951.

Among the weeds, plantain and the short and giant ragweed are the commonest offenders.

While the diagnosis of seasonal hay fever is made primarily from the history, the specific offending pollen is determined by skin-testing. In this clinic we use solutions of pollen extract for testing and treatment. The intradermal method of testing is employed. The strongest solution that we use is a 0.1 solution, which means 0.1 mg. of total nitrogen per cubic centimeter. This is diluted 1 in 10 to a 0.01 solution and again to a 0.001 solution. If necessary, a 0.0001 solution may be made. The diluting fluid is Coca's solution. It is an alkaline fluid, containing about 0.5% sodium chloride, about 0.3% sodium bicarbonate, and 0.4% phenol as preservative.

Tests are ordinarily performed with a 0.01 solution on the outer aspect of the arm by making a tiny wheal intradermally after cleansing the skin with alcohol solution. The test reactions are read at the end of about 10 min., and the patient is classified according to the severity of the skin reaction. We consider a patient as belonging to Class A if the wheal produced is about twice the size of the original wheal or larger, with definite pseudopod formation, an area of erythma, and itching. A patient is placed in Class B if the wheal produced is about twice the size of the test wheal, is round in outline, and is surrounded by an area of erythema. A Class C reaction is one less marked than the Class B type. The dose schedule which we adhere to for the various classes is as follows:

Injection	Class A		Class B		Class C	
No.	Solution	Dose, Cc.	Solution	Dose, Cc.	Solution	Dose, Cc.
1	0.001	0.1	0.001	0.1	0.001	0.3
2		0.2		0.2		0.6
3		0.3		0.4	0.01	0.1
4		0.5		0.7		0.2
5		0.7	0.01	0.1		0.4
6	0.01	0.1		0.2		0.7
7		0.15		0.4	0.1	0.1
8		0.2		0.7		0.15
9		0.3	0.1	0.1		0.2
10		0.5		0.15		0.3
11		0.7		0.2		0.4
12	0.1	0.1		0.25		0.5
13		0.1		0.3		0.6
14		0.1		0.4		0.7

While our solutions are standardized by the total nitrogen method, there are other pollen standards in use as well. At present neither the Council of Pharmacy and Chemistry of the American Medical Association nor the authorized committee of the United States Public Health Service has designated an official standard pollen unit.

The four usual methods of standardizing extracts are (1) the total nitrogen method, which we use and in which a unit equals 0.00001 mg. of total nitrogen per cubic centimeter; (2) the Noon unit, equivalent to 1/1,000,000 gm. of dry pollen; (3) the protein nitrogen (P. N.) unit, which equals 0.00001 mg. of protein nitrogen in 1 cc. of extract, and (4) the dilution standard, based on a weight-by-volume extraction and dilution; a 1:100 extract contains 0.01 gm. of extracted pollen per

cubic centimeter. From this it can be seen that 1 P. N. unit = 2 Noon units = 0.000026 mg. of total nitrogen = 2.6 total nitrogen units = 1:500,000 dilution (pollen weight by volume).

From these figures the difficulty encountered in attempting to switch from one standard to another is apparent. If one becomes acquainted with the general principles of treatment, it makes little difference which standard is used. It is best to become accustomed to a single standard and to continue its use.

No schedule can be adhered to rigidly, for often modifications are found necessary because of a variety of circumstances. Marked local or constitutional reactions, irregularity of the patient in keeping his appointments, or change of extract will interfere with the schedule.

In the case of trees, there is no cross-protection of the various pollens except within each genus. If favorable results are to be obtained, desensitization should be carried out with the individual pollen. A mixture of the extracts of several tree pollens may be used, although it should be made certain that the dose of the individual component is adequate.

In the case of grass pollen, multiple sensitivity to the various grasses is usually present, but adequate treatment with timothy pollen extract suffices to protect against the other grasses as well. Therefore, in cases of grass-pollen sensitive, we test with an extract of timothy pollen only and omit testing for the other grasses, except in some unusual cases, or if the response to treatment with timothy pollen extract is inadequate.

Within the ragweed family, group protection is the rule. Although it has been the custom to make extracts of equal parts of large and small ragweed, it was shown experimentally and clinically that an extract of either alone would suffice.

Successful treatment will depend upon several factors: the time the patient presents himself for treatment in relation to the season, the severity of the local skin reaction, and the presence of single or multiple sensitivity. At times the seasonal cases are aggravated by a summation of irritants, such as dusts or animal emanations, in addition to pollen. Food sensitivity may also aggravate the symptoms of pollinosis. In such cases food avoidance, in addition to hyposensitization, is necessary to obtain good results. The presence of infection or structural alterations in the upper respiratory tract may account for poor results.

Usually treatment is carried out by one of three plans: the prophylactic or preseasonal, the co-seasonal, and the perennial. The plan selected depends, among other things, upon the time the patient presents himself for treatment.

Preseasonal treatment is begun about three months before the onset of the hay-fever season. Injections are given once a week, according to the schedule outlined. An attempt is made to reach the maximum dose before the season begins, and treatment is stopped at that point. An alternate method is to decrease the dose to about half and continue the injections every two or three weeks through the season.

The injections can be continued every three weeks at the lower dosage through the winter on the perennial basis. As the season approaches, the time interval between injections is shortened to two weeks.

Constitutional reactions are more apt to occur in patients receiving treatment on the perennial schedule, and they should be guarded against. The interval between injections should not be longer than three or four weeks. When fresh extracts are used, it is wise to decrease the dose for one or two injections, or to mix some of the old extract with the new.

The co-seasonal method of treatment is advantageous in those patients who come too late for preseasonal therapy. The doses of pollen extract given are much smaller and are increased daily or every other day. As soon as improvement is observed, the intervals between injections are lengthened to once a week.

Good results are also obtained with the intracutaneous method of treatment co-seasonally. Very dilute solutions are used. Satisfactory relief is said to be obtained, according to Hansel, if the wheals produced by the intracutaneous injections are 20 to 25 mm. in diameter. The amount of extract injected is 0.01 to 0.05 cc. of solutions of increasing strength. A definite schedule is followed; treatment is given daily or every other day, guided by the reactions and relief of symptoms.

Each patient is an individual problem. The dose schedules usually suggested in textbooks or accompanying the packages of pollen extracts on the market should not be followed blindly. If the patient is tolerant, all well and good, but a marked local or even a mild constitutional reaction requires a reduction of the quantity of extract administered, and subsequent injections should be given with caution. The patient should not be allowed to leave the office for at least 15 to 20 min. after treatment. Solution of epinephrine (adrenalin®) and a tourniquet should always be on hand, ready to use if there is a tendency for a constitutional reaction to develop. If a reaction is imminent, the tourniquet should be applied proximal to the site of injection and about 1 cc. of epinephrine administered, a portion given at the site of injection and the remainder in the other arm. When the symptoms begin to subside, the tourniquet is intermittently loosened. If necessary the epinephrine injection may be repeated. Ephedrine sulfate, ¾ grain (50 mg.), or the antihistaminics are given in addition.

From a review of the literature, it is difficult to determine which plan of treatment gives the best results. There are advocates for each plan, with series of cases and statistics to bear out their contention. It seems reasonable, therefore, to use whichever plan is most convenient for the patient, compatible with a satisfactory result. If a good result is not obtained with one plan of treatment, change to another. In case of failure, it is wise to retest the patient and to search for additional offending substances.

It is well at this point again to emphasize that simple, uncomplicated hay fever usually responds well to pollen treatment alone. Failure to respond to specific treatment necessitates a restudy of the patient. In most instances it will be found that there are multiple sensitivities. The offending substance, in addition to the usual pollen, might be some other uncommon pollen or an inhalant of some kind, or, there may possibly be a complicating food allergy. While a single sensitizing substance acting alone may cause only negligible symptoms, if it acts in combination with other allergens the symptoms may be quite severe. If such multiple sensitivity is found, proper treatment for each of the items, either by food avoidance or by hyposensitization with the necessary inhalants, should be instituted to obtain good results.

Patients with structural defects of the nose or with sinus infections respond poorly to treatment. These abnormalities should be corrected out of season. Surgical procedures are not recommended during the season, or when the patient is receiving treatment.

The relief obtained from treatment is of a temporary character, and injections must be given each year. There is a small percentage of patients that will become symptom-free as a result of one or more years of treatment, but in the vast majority of cases symptoms will reappear if treatment is discontinued.

The antihistaminics are valuable as an adjuvant to the hyposensitization method of treatment. They do not prevent the allergic response in the body but prevent the resultant symptoms by their action of competitive inhibition in blocking histamine. Methods are being developed to integrate hyposensitization therapy with the antihistaminic treatment, so that larger doses of pollen antigen may be given with safety. In some patients to whom adequate doses could not be given hitherto this combined method will permit the administration of larger doses of pollen extract and, thus, confer a greater degree of protection, and in some instances shorten the period of treatment.

NEUROFIBROSARCOMA OF THE FACIAL NERVE INVOLVING THE TYMPANOMASTOID

M. REESE GUTTMAN, M.D.
AND
MARSHALL U. SIMON, M.D.
CHICAGO

NEOPLASMS of the intratemporal portion of the facial nerve are extremely rare. A careful search of the literature has revealed but 17 cases.[1] With the exception of a malignant neurogenic spindle-cell sarcoma reported by Kettel [1a] in the November, 1950, issue of the ARCHIVES OF OTOLARYNGOLOGY, all cases have presented the histologic picture of benign neurinoma. This report is concerned with a second case of malignant neurofibrosarcoma, that of a patient who has remained well since being observed and operated on by one of us (M. R. G.) in 1942. The rarity of the condition and the unusual life expectancy in spite of the histologic evidences of malignancy prompt this report.

Neurinomas may involve any peripheral or cranial nerve. Virchow, in his classic treatise on tumors, classified all neoplasms involving nerves or nerve tissue as neuromas. It remained for von Recklinghausen, in his histologic studies, to show that these neoplasms were derived mainly from the connective-tissue elements of the nerves and, consequently, they were simply fibromas with nerve fibers passing either through or about them. Nerve tumors originate, for the most part, from the cells of Schwann and connective tissue [2] and have no nerve-cell structure whatsoever.[3] They are benign and cause local pressure and destruction by expansile growth. They are of ectodermal origin. Less frequently growths arise from the mesodermal sheath of the nerve. These are the so-called sarcomatous variety and

---

Presented before the Chicago Laryngological and Otological Society, March 5, 1951.

From the Departments of Otolaryngology, Stritch School of Medicine, Loyola University, and Henrotin Hospital.

1. (*a*) Kettel, K.: Intratemporal Sarcoma of the Facial Nerve, Arch. Otolaryng. **52**:778 (Nov.) 1950. (*b*) Schroeder, R.: Dansk oto-laryng. selsk. forh. **31**:56, 1930. (*c*) Altmann, F.: Monatsschr. Ohrenh. **69**:1032, 1935; ibid. **71**:1287, 1937. (*d*) Bogdasarian, R. M.: Neurinoma of Facial Nerve, Arch. Otolaryng. **40**:291 (Oct.) 1944. (*e*) Lundgren, N.: Nord. med. **28**:2384, 1945. (*f*) Kettel, K.: Nord. med. (Hospitalstid.) **24**:1783, 1944. (*g*) Kos, C. M.: Ann. Otol. Rhin. & Laryng. **49**:151, 1940. (*h*) Reference deleted. (*i*) Wyosotskaja, L. D.: Ibid. **34**:326, 1941. (*j*) Roberts, G. J.: Neurinoma of Facial Nerve in Middle Ear and Mastoid: Report of Case, Arch. Otolaryng. **37**:62 (Jan.) 1943. (*k*) Williams, H. L., and Pastore, P. N.: Neurofibroma of Facial Nerve and Facial Canal: Destruction of Labyrinth and Mastoid Process, ibid. **29**:977 (June) 1939.

2. Gagel, O., in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Springer-Verlag, 1935, Vol. 11, p. 216. Verocay, J.: Beitr. path. Anat. u. allg. Path. **48**:1, 1910. Antoni, N.: Über Rückenmarkstumoren und Neurofibrome, Munich, J. F. Bergmann, 1920.

3. Loeliger, H. T. H.: Acta oto-laryng. **35**:543, 1947.

are but relatively malignant.[4] So far as the cranial nerves are concerned, these growths originate either from a sensory nerve or from a motor nerve with sensory fibers. An example of the former is the classic neurinoma, the acusticus tumor of the eighth nerve. An example of the latter, the growth which involves the sensory fibers of the facial nerve, is the subject of this presentation.

Histologically, benign neurofibroma is composed of long slender cells, with elongated nuclei, frequently arranged in palisades, whorls or eddies, this arrangement being a conspicuous and characteristic feature. At times the structure may show evidences of degeneration, when the fibrils become loose or absent, and when there are areas of round cystic spaces and hyalinization. The malignant or sarcomatous type of neurofibroma shows, in addition, irregularity in size, shape, and arrangement of the cells, as well as an increase in the cellularity of the section and a decrease in the fibrous elements.[5] While mitotic figures are infrequent, the nuclei present irregularities in size, shape and staining quality, and the tendency toward palisade and whorl formation is definitely decreased. In spite of the microscopic evidences of malignancy, these growths tend to grow slowly, remain circumscribed, and rarely metastasize. These microscopic and biologic characteristics apply to neoplasms originating from the facial nerve. When this nerve is involved, it is usually in the descending portion and less frequently in the horizontal portion.

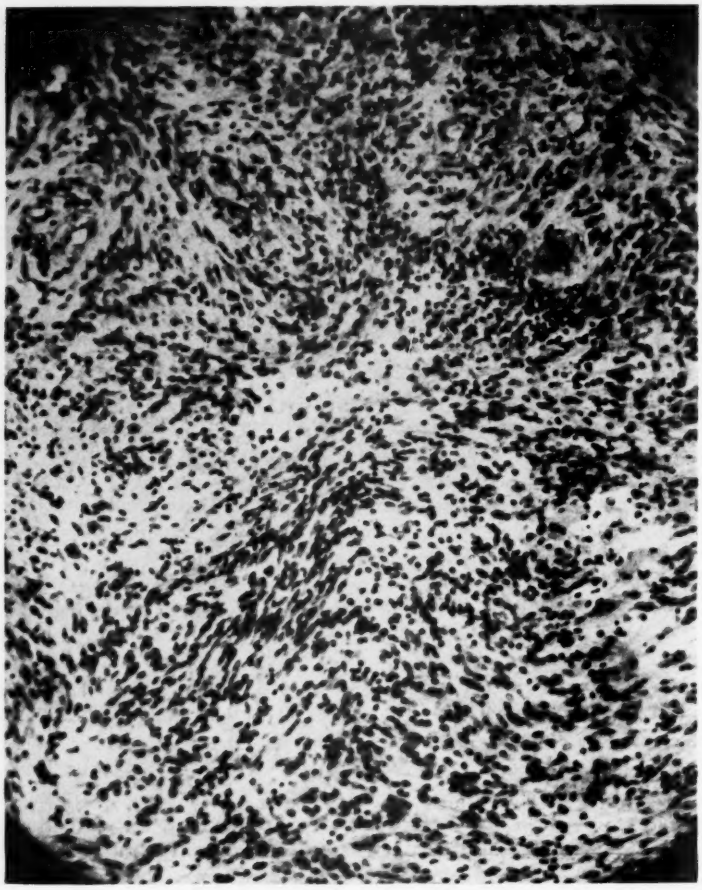
The signs and symptoms revealed by a study of the 17 cases of intratemporal facial-nerve neoplasms constitute a well differentiated clinical picture. They appear, as a rule, early in middle life. The first, and frequently the only, symptom is a peripheral type of facial paralysis, which progresses slowly over a period of months or years before becoming complete and which is not accompanied by pain or headache.[6] This pattern differentiates facial neurinoma from Bell's palsy, which comes on suddenly and is complete in a few days, and which is accompanied by severe pain in its early stages. Facial neurinoma is also differentiated from an acusticus tumor by lack of headache, tinnitus, and nerve or perception deafness, as well as lack of symptoms of dysequilibrium. Deafness comes on later in the disease and is of the conductive type due to involvement of the middle ear and tympanum. When the growth invades and destroys the tympanic membrane, otorrhea appears, owing to secondary infection. In addition, the external auditory meatus contains the neoplastic tissue, which appears not unlike granulation tissue, and is accessible for biopsy and microscopic study. The growth found at the time of surgery or at autopsy is extensive: It involves the middle ear and mastoid and, at times, the tissues about the external auditory canal; it exposes the dura of the middle and posterior fossae; and it invades the petrosa and labyrinth. In spite of such extensive involvement, intracranial complications are not very common. Carcinoma of the tympanomastoid may resemble facial neurinoma in the otorrhea and deafness, but facial-nerve tumor is distinguished by the history of an antecedent, slowly progressing facial paralysis, while facial paralysis in carcinoma comes late, after pain, deafness and otorrhea have become apparent. Inasmuch as the facial neurinoma is slow in expansion and only mildly malignant, probably many of such tumors have masqueraded under erroneous diagnoses of Bell's palsy. It is apparent, from a

4. Busch, E., and Christensen, E.: Acta psychiat. et neurol., Supp. 46, p. 72, 1947.
5. Quick, D., and Cutler, M.: Ann. Surg. **86**:810, 1927.
6. Kettel, K.: Neurinoma of Facial Nerve, Arch. Otolaryng. **44**:253 (Sept.) 1946.

consideration of the pathological and clinical characteristics, that the prognosis in cases of these growths, both benign and malignant, is good so far as life is concerned but poor so far as recovery of the facial paralysis is concerned.

REPORT OF A CASE

L. H., a 43-yr.-old white man, entered Henrotin Hospital on May 21, 1942, complaining of right facial paralysis and discharge, swelling and deafness of his right ear. The paralysis was



Neurofibrosarcoma of the facial nerve.

first noted on studying proofs of his wedding pictures of 1933, in which it was observed that his smile was unsymmetrical. The facial deformity became gradually more pronounced until finally his face was completely paralytic. Increasing difficulty in hearing on the affected side became apparent, and this was followed by a spontaneous discharge of pus from the right ear, which came on in a painless fashion. The patient had been observed by several otologists, one of whom made a diagnosis of chronic mastoiditis and performed a radical mastoidectomy in 1938, without

any observable effect on the paralysis, deafness, or discharge. Late in 1941 the patient noted a swelling about the ear which became progressively worse. His past and family history revealed nothing of significance as to his complaints.

Examination revealed a well-developed, well-nourished white man, mentally alert and cooperative. He had a complete peripheral facial paralysis, with loss of taste for sweet, sour, and salt along the right side of his tongue. There was a diffuse, soft, nontender swelling beneath and in front of the right auricle. The canal was filled with a seropurulent discharge. After the canal was cleansed of the exudate, some grayish-pink granulation tissue could be discerned. It was rather firm to the touch but bled easily. Hearing was reduced to whispered voice at contact. Tests with tuning forks and audiometer disclosed a marked deafness of the conductive type. A roentgenogram disclosed an extensive defect of the mastoid process, which was interpreted as operative in origin. A diagnosis of chronic mastoiditis and possible carcinoma of the tympanomastoid was made.

The patient was operated on on May 22, 1942. An incision was made into the preexisting postauricular scar and the soft tissues were elevated. The mastoid terrain was exposed and found to be filled with a pearly, grayish-blue, highly vascular tissue that was exceedingly friable. The mass was carefully removed until the dura of cerebrum, the cerebellum, and the lateral sinus was exposed. The tissue was firmly attached to the underlying dura and was carefully shaved down until the dura was almost reached. The mass continued down beneath the mastoid and extended anteriorly to just in front of the ear. The report on frozen section of the growth taken at the operation was neurofibrosarcoma, grade 1 or 2. The operative terrain was packed wide open, and a few days later radium was introduced, a total dose of 400 mg.-hr. being given.

The pathologist, Dr. Sam Levinson, reported that the several pieces of irregular gray tissue submitted for examination showed, histologically, a fibrillar stroma composed of elongated spindle-shaped cells, rather well differentiated. The nuclei stained deeply and were fusiform to oval in shape. In some areas the cells were arranged in bands and in others in whorls and in palisades. There were other areas in which the stroma was more fibrous than cellular, and these were dense, compact, and at times hyalinized. There were also areas of dilated blood vessels and spicules of bone both healthy and necrotic. No mitoses were seen, but the irregular size, shape and arrangement of cells suggested malignant change. The diagnosis was neurofibrosarcoma.

The patient made an uneventful recovery and was discharged from the hospital. The entire operative area healed over, and the external auditory canal closed by an epithelial membrane, although the facial paralysis and deafness remained unchanged. His condition was satisfactory until August, 1944. At that time he returned, complaining of a recurrence of the aural discharge. Examination disclosed a small granuloma in the center of the epithelial membrane occluding the canal. This was removed at the Henrotin Hospital on Aug. 7, 1944. The pathologist reported that the section was composed of fibrous tissue containing whorls simulating nerve fibers. Although from the section the growth appeared benign, the clinical history had to be taken into consideration and the diagnosis of neurofibroma was made.

The epithelial membrane healed, and the patient remained well, except for facial deformity and deafness, until June, 1946. At that time he returned, complaining of headache and impairment of vision. Examination disclosed an increase in swelling about the mastoid and bilateral papilledema. He was referred to a neurologic surgeon, Dr. Eric Oldberg, who did an exploratory operation on July 30, 1946. He found an enormous growth, involving most of the right cerebellar dura, which he removed down to the dura. The pathologist, Dr. Edwin F. Hirsch, thought that the growth was a neurofibroma and not malignant. The sections were then submitted to Dr. Percival Bailey, who agreed with Dr. Levinson that the growth was a fibrosarcoma of low grade malignancy. The patient recovered from the procedure and has been well since. His last visit, on Dec. 9, 1950, showed the healed external auditory canal, and he had no complaints other than the existing facial paralysis and deafness.

SUMMARY

Neurinoma (benign) and neurofibrosarcoma (malignant) tumors of the intratemporal portion of the facial nerve are a rare and infrequent cause of facial paralysis. They present a clinical picture that is clear and easily distinguished from other

causes of facial paralysis of the peripheral type, since they are not attended by pain, headache, and disturbances of equilibrium. They produce deafness, granulations in the canal, and otorrhea late in the course of the disease. The deafness is of the conductive type. Prognosis is good for life expectancy and poor for recovery of the facial paralysis. In addition to the case reported here, but one other in a total of 18 cases showed microscopic evidence of malignancy. In spite of this difference in the histologic picture, the clinical course of these two patients showed only insignificant variation from that of the other 16 cases.

30 N. Michigan Blvd.

USE OF A TANTALUM FOIL TUBE TO MAINTAIN PATENCY OF THE NASOFRONTAL DUCT

WILLIAM C. HUGENBERG, M.D.
AND
HENRY L. WILLIAMS, M.D.
ROCHESTER, MINN.

THE PROBLEM of maintaining the patency of the nasofrontal duct after external operations on the frontoethmoid group of sinuses is probably the most annoying confronting the rhinologic surgeon. Since the advent of operations on the frontal sinus, numerous methods and procedures have been devised and suggested to solve this difficulty. No one method to date has met general acclaim.

It was thought that lining the raw surfaces of the nasofrontal opening with skin or mucous membrane might be the proper approach. Mucosal flaps were suggested by Killian and Mithoefer. Killian [1] suggested cutting and dissecting downward to the floor of the nose a flap of mucosa from the superior turbinate before beginning the external incision. This flap is used to line the nasofrontal opening at the end of the operation. Mithoefer [2] advocated the Killian technique and suggested that in addition, if possible, a flap of mucous membrane be secured from the anteromedial wall of the frontal sinus that could be carried downward over the remaining portion of the processus frontalis. Free mucous membrane grafts were suggested by Laval,[3] who advocated the use of mucous-membrane transplants from the lower lip. The use of skin grafts to line the nasofrontal opening was suggested by Smith [4] and Howarth.[5]

Various anatomic features have been pointed out as essential to maintaining the nasofrontal opening. Anderson [6] early pointed out the necessity of doing a complete operation, particularly in regard to fashioning an adequate intranasal opening. Lynch [7] advocated wide removal of the lamina papyracea and the frontal

---

Fellow in Otolaryngology and Rhinology, Mayo Foundation (Dr. Hugenberg). From the Section on Otolaryngology and Rhinology, Mayo Clinic (Dr. Williams).

1. Killian, G.: Die Killian'sche Radicaloperation chronischer Stirnhöhleneiterungen: II. Weiteres kasuistisches Material und Zusammenfassung, Arch. Laryng. u. Rhin. **13**:59-88, 1903.

2. Mithoefer, W.: How and When to Operate upon the Ethmoid Sinuses, Surg., Gynec. & Obst. **70**:539-542 (Feb.) 1940.

3. Laval, cited by Anthony, D. H.: South. M. J. **33**:949-955 (Sept.) 1940.

4. Smith, F.: Management of Chronic Sinus Disease, Arch. Otolaryng. **19**:157-171 (Feb.) 1934.

5. Howarth, W.: Some Points in Technique of the Frontoethmoidal Operation, J. Laryng. & Otol. **51**:387-390 (June) 1936.

6. Anderson, C. M.: External Operation of the Frontal Sinus: Causes of Failure, Arch. Otolaryng. **15**:739-745 (May) 1932.

7. Lynch, R. C.: The Technic of a Radical Frontal Sinus Operation Which Has Given Me the Best Results, Laryngoscope **31**:1-5 (Jan.) 1921.

process of the maxilla. Since this seemed to promote a tendency toward closure of the nasofrontal opening, Neivert [8] advocated the use of cartilage covered with mucous membrane or skin on the medial side to replace the orbital wall removed and thereby prevent collapse of the soft tissues. Lillie and one of us (Williams) [9] have pointed out the advantage of preserving the frontal process of the maxilla, which prevents the tendency of the soft tissue to collapse into and close the nasofrontal opening. Mithoefer [2] advocated the preservation of the posterior portion of the lamina papyracea to prevent prolapse of orbital tissue. Williams and Mousel [10] urged that care be taken to approximate the cut edges of the periosteum, as this was an additional help in preventing an inward collapse of the soft tissues.

Another method of preserving a patent nasofrontal opening was suggested by Sewall.[11] He retained a portion of the lacrimal bone and frontal process of the maxilla to serve as an osteoplastic flap. Lothrop [12] suggested removing the intersinus septum and carrying the removal down into the septum nasi so as to assure drainage into the opposite nasal chamber.

It is the general opinion that the mucosa of the nasofrontal duct should be left intact whenever possible. Walsh [13] credited Mosher as being one of the first surgeons to hold this view. Begley and Hallberg,[14] in reviewing the surgical operations for osteomas of the paranasal sinuses encountered and treated at the Mayo Clinic, found that rapid healing and permanent relief of symptoms occurred in all patients in whom the frontal duct could be left intact.

The use of radium postoperatively to prevent cicatricial closure of the nasofrontal duct was suggested by Williams and Fricke,[15] and they secured partial success with this method.

Tubes of various materials placed in the nasofrontal opening have probably been used most widely. Lynch suggested the use for a short time of a rubber tube in the nasofrontal opening. Anderson [16] substituted a petrolatum-gauze pack for the tube. He suggested that it be slowly removed over a period of four to five days after the operation. Patency was then maintained by daily insertion of dilators.

---

8. Neivert, H.: Surgery of the Nose and Sinuses, in Kernan, J. D.: Surgery of the Nose and Throat, Thomas Nelson and Sons, New York, 1942, p. 192.

9. Lillie, H. I., and Williams, H. L.: The External Fronto-Ethmo-Sphenoid Operation: A Critical Review of the Literature and Details of Technic in Use at the Mayo Clinic, Minnesota Med. **18**:786-789 (Dec.) 1935.

10. Williams, H. L., and Mousel, L. H.: Diagnosis and Treatment of Chronic Disease of the Paranasal Sinuses, Ann. Otol., Rhin. & Laryng. **49**:466-489 (June) 1940.

11. Sewall, E. C.: Fronto-Ethmosphenoidectomy: Further Experience with This Operation Performed Under Local Anesthesia; A New Osteoplastic Flap, Arch. Otolaryng. **8**:144-150 (Aug.) 1928.

12. Lothrop, H. A.: Frontal Sinus Suppuration with Results of New Operative Procedure, J. A. M. A. **65**:153-160 (July 10) 1915.

13. Walsh, T. E.: Experimental Surgery of the Frontal Sinus: The Role of the Ostium and Nasofrontal Duct in Postoperative Healing, Laryngoscope **53**:75-92 (Feb.) 1943.

14. Begley, J. W., Jr., and Hallberg, O. E.: Osteomas of the Paranasal Sinuses and Their Treatment, Proc. Staff Meet., Mayo Clin. **25**:13-16 (Jan. 4) 1950.

15. Williams, H. L., and Fricke, R. E.: The Use of Radium in Maintaining a Patent Frontonasal Opening in External Operations on the Fronto-Ethmoid Group of Sinuses: A Preliminary Report, Ann. Otol. Rhin. & Laryng. **48**:412-418 (June) 1939.

16. Anderson, C. M.: The Lynch Type of Radical Frontal Sinus Operation, Minnesota Med. **11**:461-464 (July) 1928.

Erich and New [17] advocated the use of an acrylic obturator instead of a rubber tube to maintain patency of the nasofrontal duct. The use of a metallic tube to maintain a patent nasofrontal duct began with Ingals,[18] who in 1905 suggested the use of a gold tube. This was the first tube designed to remain in place for a long period. In 1940, Anthony [19] presented an excellent review of the literature pertaining to management of the nasofrontal duct. He reported the use of the Ingals tube in nine cases with very satisfactory results and later [20] mentioned a case in which a tube was in place for 18 yr. without difficulty.

Goodale [21] reported eight cases in which the use of tantalum foil as a flat strip sewn to the orbital periosteum prevented granulations from the periorbita of the medial wall from growing into and thereby closing the nasofrontal opening. Fox [22] discussed the use of tantalum in rhinoplastic surgery and presented an excellent review of the literature on the metallurgy of tantalum and its clinical uses. He reported one case in which tantalum was used according to the method of Goodale with a gratifying result. Kaplan, Schwartz, and Metson [23] reported on the use of tantalum foil and an acrylic tube, separately or in combination, in the postoperative management of mucoceles of the frontoethmoid group of sinuses.

A case was observed at the Mayo Clinic in which it was thought that a tube fashioned from tantalum foil might solve the problem of closure of the nasofrontal opening.

REPORT OF A CASE

A man aged 47 came to the clinic in June, 1947. Three months prior to this date he had had a severe head cold followed by a left maxillary sinusitis with considerable purulent discharge from the left side of the nose and pain over the left canine fossa. Five weeks before he came to the clinic, pain and swelling had developed over the left frontal region. He had been treated with local heat and penicillin injections, and three weeks before he came to the clinic a trephine opening had been made into the anterior wall of the left frontal sinus. The swelling and pain had subsided after this procedure, but a profuse purulent drainage had continued from the fistulous tract. The patient had sustained a compound fracture of the left frontal sinus 11 yr. previously, and "some bones" had been removed from the anterior wall of the sinus.

Examination of the patient at the clinic revealed a small incision over the left frontal sinus, from which a rubber drainage tube protruded. Profuse discharge of thick yellow-green pus was coming from around the tube. Edema, induration, tenderness, and erythema were present over the supraorbital region, and there was swelling of the left upper eyelid. The results of examination of the ear, nose, and throat were otherwise essentially normal, except for congested mucous membrane and pus in the left middle meatus.

The results of the general physical examination were normal. Laboratory findings were within normal limits except for a slightly elevated leukocyte count. Roentgenograms revealed

17. Erich, J. B., and New, G. B.: An Acrylic Obturator Employed in the Repair of an Obstructed Frontonasal Duct, Tr. Am. Acad. Ophth. **51**:628-632 (July-Aug.) 1947.

18. Ingals, E. F.: New Operation and Instruments for Draining the Frontal Sinus, Ann. Otol., Rhin. & Laryng. **14**:513-519 (Sept.) 1905.

19. Anthony, D. H.: Use of Ingals Gold Tube in Frontal Sinus Operations, South. M. J. **33**:949-955 (Sept.) 1940.

20. Anthony, D. H.: Personal communication to the authors.

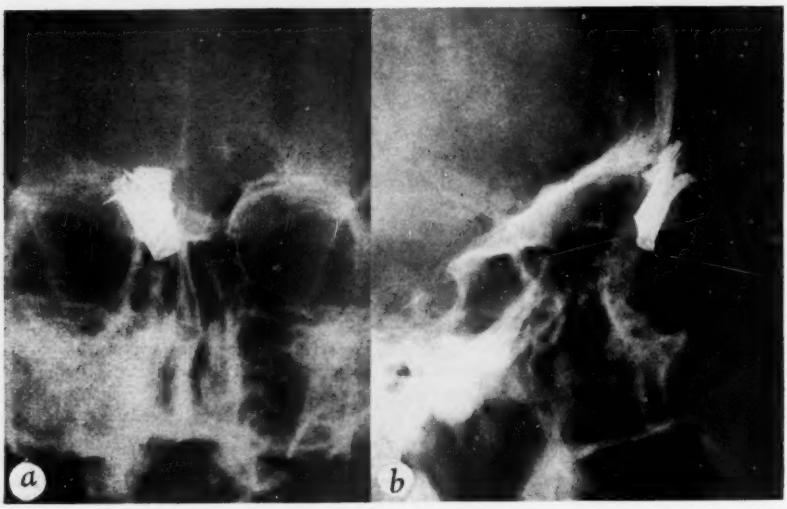
21. Goodale, R. L.: Use of Tantalum in Radical Frontal Sinus Surgery, Ann. Otol., Rhin. & Laryng. **54**:757-762 (Dec.) 1945.

22. Fox, S. L.: Tantalum in Rhinoplastic Surgery, Ann. Otol., Rhin. & Laryng. **58**:40-54 (March) 1949.

23. Kaplan, S.; Schwartz, A., and Metson, B. F.: Mucocele of the Frontal and Ethmoid Sinuses: Evaluation of the Use of Acrylics and Tantalum in the Surgical Treatment, Arch. Otolaryng. **51**:172-187 (Feb.) 1950.

diffuse opacity of the left maxillary sinus, an old fracture of the lateral border of the left orbit, thickened membrane in the left frontal and ethmoid sinuses, and marginal osteitis of the left frontal sinus. In view of the involvement of the maxillary and ethmoid sinuses on the left, it was felt that a two-stage operation should be done. Exenteration of the ethmoid cells and antrotomy by the intranasal route, followed by an ablating operation on the left frontal sinus, were suggested.

The patient proved to be too intractable to allow an intranasal operation under local anesthesia; therefore it was decided to do the whole operation by an external approach. By means of a coronal incision, the scalp was turned down over the frontal sinus, the periosteum was incised and lifted from the bone, and the fistula to the sinus exposed. The frontal sinus was opened through the anterior wall and was found to be filled with pus and thick pyogenic membrane. The sinus extended more than 1 in. (2.5 cm.) to the right of the midline. The anterior and inferior walls of the left frontal sinus were completely removed and the superior edge beveled so as to prevent "dead space." Care was taken to remove all vestige of the mucosa lining the left frontal sinus. The orbitoethmoid cells were well developed, and when



(*a*) Posteroanterior roentgenogram showing tantalum prosthesis in the nasofrontal opening on the left side; (*b*) lateral roentgenogram made in the same case.

they were exenterated an infundibuliform opening leading into the nasal chamber was left. The lamina papyracea was completely preserved, and the ethmoid cells were exenterated without removing it or the maxillary process of the frontal bone. An antrum window was made in the usual manner by the inferior meatus on the left. Convalescence was uneventful, and the patient was dismissed in two weeks.

The patient returned to the clinic for observation six months later. He said that a head cold had developed a week previously and had increased in severity, causing pain over the left maxilla, purulent nasal discharge on the left side, and swelling of the left side of the forehead. Pain also had developed in the supraorbital area on the left and had extended to the vertex. Swelling had occurred about the left eye. The patient had been taking sulfanilamide for five or six days and penicillin for three days before he came to the clinic.

Examination revealed tenderness over the nasion and some boggy swelling over the left eye, with periorbital edema and redness. Nasal examination revealed markedly injected mucosa on the left and considerable mucopurulent discharge. The nasofrontal duct could not be probed after shrinkage.

Roentgenograms revealed postoperative changes in the left frontal area with diffuse opacity of the left frontal sinus, left ethmoid cells, and left maxillary sinus.

It was felt that the patient had a subperiosteal abscess in the left nasofrontal region.

The left frontal sinus was opened through the brow. A large abscess was entered which had elevated the periosteum over the frontal area of the ablated sinus. There was no evidence of cyst formation from incomplete removal of the mucosa. The recurrence was thought to be due to closure of the nasofrontal opening so that a pocket had formed in the infundibuliform entrance and had later dissected beneath the periosteum over the frontal bone. A 2 by 3 cm. piece of tantalum foil was coiled and then bent to conform to the nasofrontal opening and fixed in place by flanging the upper end. This is best seen in the accompanying roentgenograms (Fig.). The incision in the brow was closed in the usual manner.

The patient returned for observation two years later. Pain and tenderness had developed over the left eye in the region of the external frontal scar a week previously. The pain had continued and had become localized in the region of the supraorbital nerve.

Examination revealed a small swelling in the left brow, about 1 cm. wide, and slightly tender. Nasal examination revealed a small amount of pus in the ethmoid area and a mass of granulations and scar tissue about the nasal end of the tantalum prosthesis completely blocking the tube. Roentgenograms revealed the tantalum-foil tube in the frontoethmoid area with bony sclerosis in the lateral frontoethmoid area.

Because of the apparent foreign-body reaction, it was felt that the tube should be removed. An incision was made through the frontal scar. It was found that there was a small amount of pus around the upper end of the tantalum cone which had been placed there two years previously and which apparently had produced some reaction. After removal of the tantalum cone it was found that the infundibulum-shaped frontal area had been completely covered by epithelium. The epithelium was not disturbed, and the incision was closed. The patient has remained free of symptoms since the operation.

COMMENT

The reason for reporting this case is the fact that the tantalum prosthesis employed to maintain the nasofrontal opening had apparently produced a foreign-body reaction in the tissues of the nose. The result obtained is in contrast with reports in the literature that tantalum causes no noticeable reaction of adjacent tissues. It would seem from our experience that this metal had produced a foreign-body reaction sufficient to block the tube and necessitate its removal.

SUMMARY

In a case in which tantalum foil was employed to maintain the patency of the nasofrontal duct, the result indicated that tantalum is not as inert in body tissues as could be desired.

Case Reports

CONGENITAL NEUROGENIC MALFORMATION OF THE FAUCIAL TONSIL

G. KENNETH LEWIS, M.D.
WERNER K. GOTTSTEIN, M.D.
AND
VINCENT A. COSTANZO Jr., M.D.
CHICAGO

TONSILLAR growths, particularly those of congenital origin, form a highly controversial problem. It is the consensus among investigators that congenital tumors are very rare and, as a search of the literature reveals, are omitted from many comprehensive works on pathology.

Many congenital tumors once described as growths have later been interpreted as hyperplasia of normal tissue. A few cases of teratomas and dermoid cysts have been described but not sufficiently analyzed from the histological point of view. Blair,[1] however, points to the possibility that congenital tumors are of fairly frequent occurrence but, being easily detached, are often lost without detection.

Meyer [2] and Grunwald [2a] have demonstrated in chicken and human embryos the relationship between the formation of the notochord and the tissues of the primitive pharynx. Since the faucial tonsil results from an infiltration of lymphocytes into the dorsolateral wall of the second pharyngeal pouch, the invasion of neurogenic tissue is adequately explained by this early relationship. Embryologically, two median dorsal tubular evaginations are closely associated with the pharyngeal membrane, one on its ectodermal border, the other on the entodermal border. The ectodermal tubule in the roof of the oral sinus is known as Rathke's pouch and represents the anterior lobe of the future hypophysis cerebri. The shallower entodermal evagination on the cephalic tip of the primitive pharynx is known as Seessel's pocket. To its apex is frequently attached the attenuated cephalic terminus of the notochord. The bursa pharyngea and Seessel's pocket are commonly regarded as identical. Huber [3] has shown, however, that in human embryos they are distinct structures. The pharyngeal tonsil develops by lymphocytic infiltration, rostral and lateral, of the pharyngeal bursa, early in the fourth month.

Finder,[4] in describing a fibroadenoma of the tonsil, emphasizes "that the benign tumors in comparison to the malignant ones, as the increasing literature demonstrates, are not infrequently localized here and decidedly belong to rarities."

1. Blair, V. P.: Tumors of the Mouth, Surg., Gynec. & Obst. **22**:117, 1916.

2. Meyer, R.: Über die Bildung: Recessus pharyngeus medius S. Bursa pharyngea in Zusammenhang mit der Chorda bei menschlichen Embryonin, Anat. Anz. **37**:449, 1910.

2a. Grunwald, L.: Eine Cyste der Chordascheide, Anat. Anz. **37**:294, 1910.

3. Huber, G. C.: The Relation of the Chorda Dorsalis to the Anlage of the Pharyngeal Bursa or Median Pharyngeal Recess, Anat. Rec. **6**:373, 1912.

4. Finder, G.: Zur pathologischen Anatomie der Tonsille, Arch. Laryng. u. Rhin. **8**:354, 1898.

Avellis [5] in a publication on tonsillar lipoma remarks "that the benign tumors of the tonsils are such rarities that they deserve some attention for this very reason." Albert [6] in describing papillomas localized in the tonsillar region expresses his

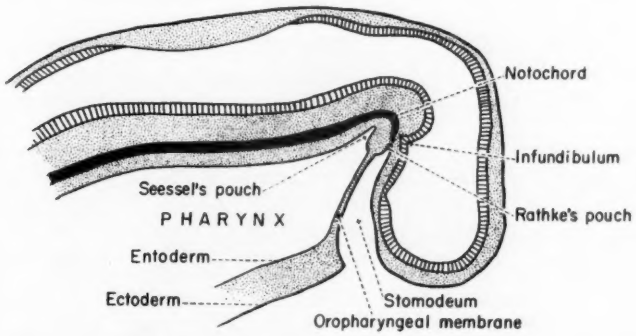


Fig. 1.—Semidiagrammatic drawing of a median longitudinal section of the head end of an embryo at the stage immediately preceding the rupture of the pharyngeal membrane; basic features redrawn from Jordan, H. E., and Kindred, J. E.: Textbook of Embryology, ed. 5, New York, D. Appleton-Century-Crofts Company, Inc., 1948, p. 114.

Fig. 2.—Tumor coming from mouth of infant.

opinion in a similar way. Lange [7] has described a case of multiple papilloma of the tonsil, epiglottis, and tongue. Fibromas of the tonsils have been described by

5. Avellis, G.: Lipom der Mandel, Arch. Laryng. u. Rhin. **8**:560, 1898.

6. Albert, E.: Einige seltenere Erkrangkungen der Zunge, Wein. med. Presse **26**:1, 1885.

7. Lange, F.: Ein Fall von multiplen Papillomen an Tonsille, Zunge und Epiglottis: Ein Beitrag zum Wachsthum der entzündlichen Papillome, Deutsches Arch. klin. Med. **1**:463, 1892.

Chiari,[8] Buhl,[9] and Prota,[10] fibroma telangiectatic by Chauveau,[11] adenofibroma by Koch,[12] and fibromatous tonsillar polyp by Midowski (angiofibroma). Biaggi,[13] Onodi,[14] Avellis,[5] Polyák,[15] and Theisen [16] have reported on lipoma of the tonsil. Stoerk,[17] Hartman,[18] and Martuscelli [19] have reported on angiomas of the tonsil.

Fig. 3.—Low-power section of cut surface of tumor.

8. Chiari, O.: Die Krankheiten des Rachens, Leipzig, F. Deuticke, 1903.
9. Buhl: Fibroerer Tumor der Tonsilla, Danischer otolaryngologischer Verein, 1900.
10. Prota, G.: Fibroma della tonsilla, Arch. ital. laring. **21**:1, 1901.
11. Chauveau: Polype de l'amygdale, Arch. therap., Sept., 1899; cited by Glas, E.: Beiträge zur Pathologie gutartiger Tonsillartumoren, Arch. path. Anat. **182**:91, 1905.
12. Koch: Ueber einen Fall von Mandelpolyp, 21, Sektion der Naturforscher, Versammlung zu Koeln, 1898.
13. Biaggi, C.: Lipoma della loggia tonsillare, Gior. 1st Nicolai, 1893, no. 1, p. 29.
14. Onodi, A.: Lipom der Tonsille, Monatsschr. Ohrenh. **29**:76, 1895.
15. Polyák, L.: Lipom der access. Tonsille, Demonstration in Gesellschaft ungarischer Kehlkopfärzte, November, 1899.
16. Theisen, C. F.: A Case of Lipoma of the Tonsil, Albany M. Ann. **24**:437, 1903.
17. Stoerk, K.: Angioma cavernosum der Mandel, Demonstration, Wiener laryng. Gesellschaft, April 1895.

Klestadt [20] states that in the majority of so-called benign tumors of the tonsils, "the true tumorous nature has not been exactly proven. Yet, beyond any doubt, only the rare teratoids, dermoids, and the simpler mixed tumors as well as the even less frequent hemangiomas and neurinomas occur." Harper [21] describes a tumor developing in and under the tonsil, extending as far into the skin of the neck as a dermoid cyst. Unfortunately, it is not described in detail.

Because of the wide divergence of opinion regarding the rarity of congenital tumors and the scarcity of reported cases in the literature, a detailed description of

Fig. 4.—High-power view of tumor.

the rare occurrence of a congenital neurogenic malformation of the faucial tonsil in a newborn infant observed by us will seem of particular interest to the embryologist and the neurologist.

18. Hartman, J. H.: A Case of Angioma of the Tonsil, with Recurrence of the Same 3 Years After Removal, New York M. J. **66**:871, 1897.

19. Martuscelli, G.: Angioma papillomatoso ed angioma semplice delle tonsille palatine, Arch. ital. laring. **21**:10, 1901.

20. Klestadt, W.: Zur Kenntnis der gutartigen Tumoren der Gaumenmandel und Bemerkungen über die Mandelkapsel, Ztschr. Laryng., Rhin. **14**:47, 1926.

21. Harper, J.: Dermoid of the Tonsil, J. Laryng. **39**:157, 1924.

REPORT OF A CASE

The tumor of the faucial tonsil which we observed was in a female newborn infant. The family history was noncontributory, and one previous pregnancy of the mother in 1948 was normal. This full-term infant was born on Jan. 8, 1950, after a labor lasting four hours. The presentation was vertex occiput anterior, the birth weight 9 lb. 7 oz. (4,280.78 gm.) and the height 21 in. (53.34 cm.). Careful physical examination failed to reveal any abnormality except this congenital tonsillar malformation.

Immediately after birth, a growth about 9 by 6 by 6 cm. was noticed hanging out of the mouth of the infant, attached by a narrow pedicle to the left tonsil fossa. It was potato-shaped, dark red in color, with areas appearing almost ready to bleed and was hard and resistant to cutting. The cut surface presented a homogeneous appearance with a few cavities filled with a serous colorless fluid and varying in size from 0.25 cm. to 2 cm. in diameter. The growth was removed by doubly clamping the pedicle at the proximal end and cutting between the clamps. The stump was then ligated and buried under the mucous membrane. The infant's recovery was uneventful.

The tumor specimen, as described by Dr. J. J. Moore, pathologist, consisted of a lobulated and encapsulated mass measuring 8.5 cm. by 6.2 cm. by 4.5 cm. It was soft and translucent and on section found to be composed of numerous cavities lined with smooth grey tissue. The wall was light greyish tan and soft and contained blood-tinged liquid. Several sections from various portions of the tumor were studied. These revealed varying tissue components. Several areas were composed of vascular nervous tissue resembling cerebral cortex. Other portions were composed of nerve tissue but had palisading of nuclei similar to the cerebellum. Some areas also were composed largely of blood vessels. One section had squamous epithelial covering beneath which was fibroadipose tissue and nerve tissue. Some cartilage and bone were present as well as glands lined by columnar and cuboidal epithelium.

SUMMARY

The rare occurrence of a congenital malformation of the faucial tonsil of neurogenic origin in a newborn infant is reported, with a possible explanation for its formation.

A detailed description of the tumor with illustrative figures is presented, with a biopsy report made after successful removal.

6 N. Michigan Ave. (2).

PARESTHESIA OF THE PHARYNX DUE TO CERVICAL ADENITIS

I. M. SCHNEE, M.D.
PATERSON, N. J.

PARESTHESIA of the pharynx induces a variety of signs, such as soreness, dryness, burning, choking, scraping, tickling, desire to swallow, sensation of a foreign body, heat, cold, and itching. There may be spasm of the larynx resulting in dysphonia or spasm of the esophagus resulting in dysphagia. These cases of paresthesia of the pharynx are usually a challenge to the examiner, for in most of them no organic basis can be found.

Diagnosis is made by a process of elimination. First, tuberculosis and tumors should be ruled out. Then, one needs to consider the numerous conditions given in the literature as possible causes, such as hysteria, neurosis, anemia, gout, dyspepsia, alcoholism, hypochondriasis, and menopause. There may be regional disease such as nasal and sinus infection, inflammation of any of the components of Waldeyer's ring, e. g., tonsils, adenoids, lateral bands, posterior-pharyngeal wall lymphoid tissue, and lingual tonsils, and ossification of the stylohyoid ligament. Reflex causes have been reported, such as cerumen or a foreign body in the external auditory canal, refractive errors of the eye, septic teeth, and disorders of the gastrointestinal tract, pelvic organs, heart, lungs, and kidneys. One must guard against attributing the cause to a local, trifling condition.

In the case I am reporting, I found the causative factor to be cervical lymphadenitis in which the node was impinging on the superior laryngeal nerve. The paresthesia cleared up after this abnormality was removed.

REPORT OF A CASE

The patient, a white 28-yr.-old man, was first seen in our clinic March 15, 1949. He stated that in 1945, after several attacks of sore throat, swallowing difficulty developed. This he described as a tightening of the throat in the course of swallowing, which was followed by regurgitation of the food into the mouth. He finally swallowed the food. In addition, he constantly had the feeling of a lump in the throat and felt pain in the right side of the neck. In 1946, a tonsillectomy was performed in the hope of effecting a cure, but there was no improvement and he still had the same complaints.

Examination revealed that the nose and paranasal sinuses were normal, the nasopharynx contained some adenoid tissue, and the oropharynx was normal. The tonsils were absent, the fossae were clear, the hypopharynx was normal, the epiglottis was overhanging, and the larynx was normal. On palpation, a tender spot was located in the right side of the neck above the hyoid bone in the region of the superior laryngeal nerve. Roentgenograms of the chest and the esophagus after the patient swallowed barium showed nothing significant. Endoscopy revealed no abnormality. Psychiatric and neurological consultation yielded no helpful information.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Since the only significant finding was the tenderness over the right superior laryngeal nerve, 1% procaine solution was injected into the latter. Immediate relief of the symptoms was obtained and lasted three weeks, after which the symptoms returned. On the basis of this experience sectioning of the superior laryngeal nerve was considered, but it was decided to observe the patient for a period of time. However, having had relief after the injection, the patient demanded that the proposed operation be performed immediately.

Thus, on June 17, 1949, an operation was performed for the purpose of sectioning the nerve. The customary incision for exposing the superior laryngeal nerve was made. In the site where the nerve is usually found, a lymph node was encountered firmly adherent to the vascular and nervous structures. The node was removed intact, and a segment of nerve and vascular structures along with it. The wound was closed without drainage and healing was satisfactory. For a few weeks postoperatively the patient had some numbness and induration in the operative area. A course of short-wave diathermy was given. One week after the operation his only complaint was slight tenderness in the operative area, and this cleared up in a few weeks. The swallowing difficulty and the other paresthesia manifestations disappeared, and there has been no recurrence to date, two years postoperatively.

Microscopic examination of the specimen removed at the operation showed a lymph node having the characteristics of chronic inflammation, muscle tissue, nerve, and blood vessels.

SUMMARY

1. A case of pharyngeal paresthesia is presented.

2. Relief was obtained temporarily by injection of the superior laryngeal nerve, and cure was obtained by sectioning the nerve and removing the inflamed lymph node that was found adherent to the nerve.

586 E. 25th St. (4).

BACILLUS PROTEUS ABSCESS OF CEREBELLAR LOBE OF OTOGENIC ORIGIN

Report of a Case with Recovery

WILLIAM R. CHAMBERS, M.D.
AND
WILLIAM CLARK, M.D.
SOUTH BEND, IND.

ACCORDING to Medwick, Uihlein, and Hallberg,[1] "cerebellar abscess is apparently a rare complication of otitis media." They reported only six cases found at the Mayo Clinic over a 10-yr. period. They also noted that Asherson,[2] in a review of the English literature in 1948, found only 41 cases of abscess of the cerebellar lobe secondary to chronic or acute otorrhea.

On the other hand, cases of proved brain abscess in any location due to Bacillus proteus are reported as rarely in the literature as abscess due to otitis media, although it appears to be the belief of some pathologists long in the field that this infectious agent has been recognized far oftener than reported.[3] T. L. McKee,[4] in a review of the literature in 1944, found reports of only four cases of brain abscess due to B. proteus. Paine, Murray, Harris, and Finland,[5] in 1947, reported one case, while Pulaski and Matthews,[6] also in 147, reported on eight brain abscesses due to gram-negative bacilli which responded to streptomycin and of which some may have been due to B. proteus.

Recovery from either an abscess of the cerebellar lobe of otogenic origin or from any abscess due to B. proteus has not been too frequent. As reported by Medwick, Uihlein, and Hallberg, some clinics have had a mortality of almost 100%. Asherson, in his review of cerebellar abscess in general, found that 41% of the patients had died. McKee reported a fatality of 50% in his four cases.

It was felt, therefore, that it might be of some interest to report a case which not only represented a cerebellar abscess of otogenic origin but also such an abscess with B. proteus as the infectious agent, making the fact of recovery doubly arresting.

---

1. Medwick, J. X.; Uihlein, A., and Hallberg, O.: Abscess of the Cerebellar Lobe of Otogenic Origin, Arch. Otolaryng. **50**:429 (Oct.) 1949.

2. Asherson, N.: Cerebellar Abscess, Acute, Otogenic, J. Laryng. & Otol. **62**:278, 1948.

3. Giordano, A. S.: Personal communication to the authors.

4. McKee, T. L.: Bacillus Proteus Infections: Review of Literature; Report of Case of Septicemia of Otitic Origin Treated with Sulfapyridine, with Recovery, Arch. Otolaryng. **39**:398 (May) 1944.

5. Paine, T. F.; Murray, R.; Harris, H. W., and Finland, M.: Streptomycin in the Treatment of Certain Gram-Negative Bacillus Infections of the Central Nervous System, Am. J. M. Sc. **213**:676, 1947.

6. Pulaski, E. J., and Matthews, C. S.: Streptomycin in Surgical Infections: Otitis Externa, Otitis Media, Mastoiditis, Brain Abscess and Meningitis, Arch. Otolaryng. **45**:503 (May) 1947.

REPORT OF CASE

The patient, a 16-yr.-old white boy, fell and struck his head in a skating accident. He was not unconscious and appeared to have suffered no injury of consequence. The next day he complained of severe hemicrania of the left side not confined to any particular region. The second day he began to vomit and was hospitalized. He rapidly improved, and after two days in the hospital, during which time roentgenograms of the skull showed no sign of fracture, he was allowed to return home.

The roentgenograms did, however, reveal a clouding of the left mastoid. This was thought not remarkable, for the patient had had chronic otitis of the left ear, intermittently draining, for six to eight years. This had been treated elsewhere with penicillin injections but never by operation.

Nine days after the injury, the headache again became severe, drowsiness appeared, and double vision was experienced. Stiff neck was also a prominent feature and vomiting recurred. Examination on the 10th day, Oct. 23, 1949, revealed an acutely ill and somewhat uncooperative and irritable boy who held his eyes closed by preference and resisted movement of his head, saying his headache was aggravated thereby.

The temperature was 96.6 F., pulse rate 46, respiration rate 16, and blood pressure 94/62. Consciousness was slightly impaired, the responses being somewhat slow. There was no obvious impairment of the external eye movements, but the fundi showed blurring of the disk margins without measurable elevation of the nerve head. There was marked nystagmus on looking to the left and slight nystagmus on looking to the right. The pupils reacted somewhat sluggishly to light; they were equal in size. The voice was slightly husky but not so much as to be obviously altered. Otherwise, the cranial nerves were intact. There was ataxia with a tendency to fall to the left, past pointing on the left, and some dysdiadokokinesis on the left. The muscle tone was not noticed to be remarkable at this time, nor were the check movements thought to be impaired. Spinal tap showed a pressure of 580 mm. of water, with a white blood cell count of 80, predominantly polymorphonuclear leukocytic, and a red blood cell count of 10. The red blood cell count was 4,890,000, the white blood count 16,300 with 85% polymorphonuclear leukocytes. The urine was not remarkable.

Within the first few hours, marked fluctuations of blood pressure up to 170/110 were noted, the pulse and respiration rates remaining normal to slow, with irregularity in rhythm. The patient then became very restless, complained of palpitation and orthopnea. Intravenous injection of concentrated dextrose temporarily improved his symptoms. The white blood cell count meantime rose to 18,400, with 87% polymorphonuclear leukocytes.

A diagnosis of cerebellar abscess of the left side was made, and an emergency suboccipital craniotomy with intratracheal ether anesthesia was performed the next day. Spontaneous respiration ceased during the approach to the occipital plate, and artificial respiration was maintained for 10 min. When the first trephine opening was made, the dura was seen to be gray-green and tense and apparently thickened. When the dura was opened, thick green-white malodorous pus welled up from beneath, under pressure. With the release of pressure, spontaneous breathing was resumed. The cerebellum beneath was soft and necrotic, and a tract led down into the left lobe to a new accumulation of pus, an intralobar abscess.

The rim of the foramen magnum and a large segment of the occipital plate were removed for decompression and a drain inserted. Culture of the purulent material yielded a predominant growth of gram-negative bacillus, further identified as B. proteus, and a smaller percentage of nonhemolytic Staphylococcus albus colonies.

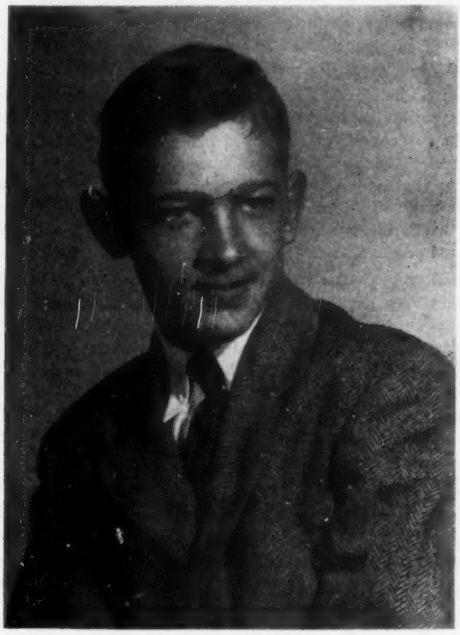
On October 23 use of streptomycin, 0.5 gm. twice a day, and penicillin, 50,000 units every three hours, was begun. On October 24, administration of aureomycin, 100 mg. intravenously every six hours, was started, and on October 25 streptomycin was discontinued.

It is interesting to note that the case of temporal abscess due to B. proteus reported by Dearmin and Sims [7] in 1940 also showed slow pulse rate, stiff neck,

7. Dearmin, R. M., and Sims, J. L.: Thrombosis of the Lateral Sinus and Abscess of the Temporal Lobe Due to Bacillus Proteus, Arch. Otolaryng. **32**:1098 (Dec.) 1940.

lateral nystagmus, and leukocytosis. Neter and Chait,[8] in 1940, reported a patient with a subdural abscess who also had a stiff neck. However, most spinal-fluid cell counts have been higher than the one recorded by them in their report. Contrary to the conclusions of Asherson,[2] who found subtentorial abscess to be associated with low spinal-fluid pressure, this patient's pressure was very high. He also noted, however, the characteristic nuchal rigidity, nystagmus, and dysdiadokokinesis.

The patient's condition postoperatively at first improved, but later marked ataxia, positive Brun's sign, difficulty in swallowing, and slight cyanosis appeared. The sensorium was moderately clouded. There was a very low grade temperature, the pulse and respiration were within the normal rate but again irregular, and the blood pressure was in the neighborhood of



The patient six months postoperatively.

135/60. A large, fluctuant, tender area was now noted in the left mastoid region, and the left ear had begun to drain. On October 28 a subperiosteal abscess over the left mastoid was drained under local anesthesia, as the condition of the patient at that time did not warrant more extensive surgery, and 3 to 4 cc. of pus was evacuated.

On October 29, the report of B. proteus infection having been returned, use of aureomycin was discontinued and streptomycin resumed, 0.5 gm. twice a day. Administration of chloramphenicol was also begun in dosage of 250 mg. every four hours, but the patient found this hard to retain. Frequent blood transfusions were given.

On October 31 improvement in his general condition permitted a left radical mastoidectomy, which disclosed extensive softening and a fistulous tract leading from the mastoid cavity into the posterior cranial fossa. The lateral sinus was found to be thrombosed and was opened wide

8. Neter, E., and Chait, R. A.: Bacillus Proteus in Otitis Media, Complicated by Mastoiditis, Meningitis and Bacteremia, Arch. Otolaryng. **32**:947 (Nov.) 1940.

until free bleeding occurred, then packed. After this operation, the dysphagia disappeared, and the diplopia subsided, but the frontal headache persisted. The vertigo, ataxia, and dysdiadokokinesis, however, progressed and the nystagmus was still present and violent. There was profuse malodorous drainage, the white blood cell count was now 11,000.

On November 5 the posterior fossa was reopened and a reaccumulation of thick pus extending well down into the left cerebellar hemisphere was found. A biopsy of the cerebellar substance was made, and the specimen was found to be so necrotic that the microscopic structure was almost entirely obliterated. A Mosher drain was inserted and removed on November 14.

On November 23 the patient suddenly became rather weak, with mottled cyanosis of the extremities, irregular respiration, and variable pulse rate; consequently, on November 25 a ventriculogram was done to rule out the possibility of other abscesses. The ventriculogram revealed nothing. The ventricular fluid did not show increased pressure.

On November 27 the Mosher drain was again inserted and kept in for 23 days. Gradually the patient improved. There was a small leak of spinal fluid at the upper angle of the wound which gradually sealed, and green drainage continued only from the ear.

The patient, on discharge Jan. 8, 1950, after 78 days in the hospital and after receiving 53.5 gm. of streptomycin, as well as 17,350,000 units of penicillin, 2,000 mg. of aureomycin, 55,750 mg. of chloramphenicol, 3.5 gm. of sulfadiazine, and 4 gm. of eskadiazine® (an aqueous suspension, 10% W/V of microcrystallin sulfadiazine in a 30 cc. solution), had a normal temperature, and other vital signs were normal. He could walk with assistance; the nystagmus was still present but mild. The most marked finding was a flail-like hypotonia of the left side of the body in which the strength was within normal limits but dysdiadokokinesis, past pointing, asynergia, and pendulant reflexes were prominent.

Twenty-six days after discharge he was again examined. The reflexes on the left side were hypoactive, the left knee had a pendulous reflex, and there was diminished muscle tone on the left. There was unsustained nystagmus on looking to the left, but the ocular disks were sharp and no papilledema was found. Marked dysdiadokokinesis and past pointing could still be noticed, and a small amount of bloody drainage from the wound continued. Two months after discharge the patient was again seen, at which time he announced he had been riding a bicycle without difficulty. The wounds were well healed and all the signs had improved.

SUMMARY

A case of cerebellar abscess and subdural abscess in the posterior fossa, of otogenic origin and due to B. proteus infection, has been presented. Cerebellar abscess from this source is rare, as is any brain abscess due to this infectious agent. Recovery is doubly remarkable since prognosis, in either of these circumstances, has been shown to have been poor in the past.

706 Odd Fellows Bldg.

120 N. Lafayette Blvd.

DERMOID CYST OF THE MIDLINE OF THE NECK

CHARLES F. GRABSKE Jr., M.D.
PHILADELPHIA

DERMOID cysts have been reported frequently in the American and British medical literature, but those occurring in the midline of the neck have been rarely mentioned. Lahey [1] reported their occurrence in an article on tumors of the neck. Papers by Clute and Cattell [2] and McClintock [3] stress the difficulty of differential diagnosis between midline dermoids and thyroglossal-duct cysts, but do not contain presentations of cases. New and Erich,[4] in a report published in 1937, clarified the classification of the three types of dermoid cysts. The cysts are subdivided into groups on the basis of their pathogenesis as well as their gross and microscopic appearance. The first type is the congenital dermoid cyst, which is teratoma-like and arises from the embryonic germinal epithelium. This is the most complex in structure and is most frequently found in the ovary and the testicle. The second type is the acquired implantation dermoid, which is caused by traumatic implantation of epidermoid tissue and is usually found on the hands and other exposed parts of the body. The third type, with which this report is concerned, is the congenital-inclusion dermoid cyst, which develops along the lines of embryonic fusion.

Arey [5] has stated that the second, third, and fifth branchial arches are the forerunners of the epidermis of the neck. The upper neck is from the second arch, the middle neck from the third, and the lower neck from the fifth. Theoretically, therefore, congenital inclusion dermoids may occur at the point of fusion of any of these structures. The cysts may arise from the infolding of a skin rudiment in the closure of any embryonic fissure or from displacement of the epithelium during the developmental period. The walls are therefore lined by stratified epithelium of the epidermoid type and may have their associated hair follicles, sweat glands, and sebaceous glands. The fluid material in the cysts is produced by these glands.

New and Erich's report [6] from Rochester of 100 cysts of the head and neck seen between 1910 and 1935 indicates that only seven were found in the cervical region and only one of these appeared to be in the midline. The pathological picture of

Read before the meeting of the College of Physicians of Philadelphia, Section on Otolaryngology, Nov. 16, 1949.

1. Lahey, F. H.: Tumors of the Neck, J. A. M. A. **138**:264-274 (Sept. 25) 1948.
2. Clute, H. M., and Cattell, R. B.: Tumors of the Neck, Ann. Surg. **92**:57, 1930.
3. McClintock, J. C.: Lesions of the Thyroglossal Tract, Arch. Surg. **33**:890-894 (Nov.) 1936.
4. New, G. B., and Erich, J. B.: Dermoid Cysts of the Head and Neck, Surg., Gynec. & Obst. **65**:48-55 (July) 1937.
5. Arey, L. B.: Developmental Anatomy: A Textbook and Laboratory Manual of Embryology, ed. 4, Philadelphia, W. B. Saunders Company, 1940.
6. New and Erich.[4]

inclusion dermoids is usually simple. They are unilocular. The capsules are composed of skin elements with the horny layer being innermost. They may contain one or all of the dermal appendages listed above. The contents are liquid or cheesy, white or yellow, and are odorless unless infected. They may shade over into a teratoma and also contain hair and bone.

The problem of differential diagnosis of infrahyoid-midline neck cysts is primarily concerned with the difference between a thyroglossal-duct cyst and a dermoid. Christopher [7] states in an article describing dermoid cysts of the floor of the mouth that the dermoid is doughier on palpation. They both, however, are fluctuant, and the skin moves easily over them. Size varies in both from a few millimeters to several centimeters. Dermoids may not move with deglutition, but since they are

Fig. 1.—Photograph of the neck showing tumor.

usually connected with deep fascia this characteristic may be misleading. Lipomas, hemangiomas, and discrete tuberculous glands must also be considered in differential diagnosis of tumors in this location but should be easily differentiated by their appearance and structure. Carcinoma of the larynx with extension into the neck may present a similar appearance but may be easily differentiated by examination of the larynx.

REPORT OF A CASE

Mr. S., aged 59, was admitted to the hospital on July 14, 1948, with the chief complaint of a mass under the chin. He had noticed the tumor about six years before and had thought it was a "double chin." About eight months before admission he was seen by a physician who had

7. Christopher, F.: A Textbook of Surgery, ed. 5, Philadelphia, W. B. Saunders Company, 1949.

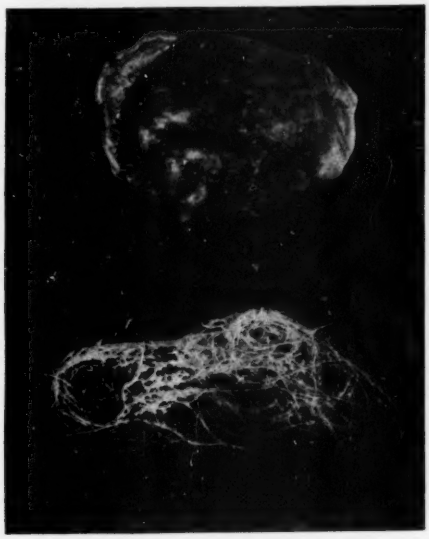


Fig. 2.—Top—excised mass; bottom—hair from the cyst.

Fig. 3.—Squamous-cell epithelium at upper left. Foam cells and granulation tissue throughout most of the section. Sweat-gland duct at lower center.

advised him to see a consultant. The advice was not immediately carried out, and the size continued to increase slowly. The mass was not painful and did not interfere with deglutition.

There was a large, rounded, fairly symmetrical swelling in the submental region. The major portion appeared to be over the hyoid bone and the upper part of the thyroid cartilage. It was in the midline and was fluctuant and elastic on palpation. The overlying skin was freely movable, and the mass was rather firmly attached to the underlying structures. It moved upward when the patient swallowed.

The mass was removed by surgical excision the following day (July 15). Under local anesthesia a transverse incision was made over the front of the neck incising the skin and platysma. The mass was fluctuant and enclosed by a thick capsule. The surrounding structures were fairly intimately adherent to the musculature but free of the hyoid bone. During the course of the separation of the sac the cyst was ruptured, and thick yellow material as well as a large mass of colorless intertwined hair was evacuated. Removal of the remaining part of the sac was accomplished without difficulty. The wound healed satisfactorily, and there has been no recurrence.

The cyst measured 6 cm. in length and 4 cm. at its greatest diameter. The microscopic section consisted of two pieces of cyst wall partly lined by stratified squamous epithelium. The rest of the lining had been replaced by numerous large foam cells and granulation tissue beneath the lining. The tissue was composed of a vascular supportive connective tissue. A small duct lined with transitional epithelium was present in the supportive connective tissue beneath the lining and probably represented a duct of a sweat gland. In other areas of the same section there were several collections of large pale foam cells with small pyknotic nuclei which were interpreted as being remnants of sebaceous glands. The diagnosis was dermoid cyst.

SUMMARY

A review of the characteristics of dermoid cysts is presented, the problems of differential diagnosis of midline-neck tumors is discussed, and a case of a midline dermoid cyst of the neck is reported.

5908 Green Street (44).

SQUAMOUS CELL CARCINOMA SECONDARY TO CHOLESTEATOMA

EDWIN H. COACHMAN, M.D.†
MUSKOGEE, OKLA.

THE FOLLOWING report is of a case of squamous cell carcinoma found in the mastoid antrum and middle area which had developed secondarily from cholesteatoma of the middle ear.

REPORT OF A CASE

R. C. C., a 38-yr.-old man, was seen on Jan. 10, 1950, with an exposed cholesteatoma protruding through a fistulous erosion (1 cm. in diameter) which had perforated the outer table of bone and the skin approximately 1 cm. posterior to the antrum area of the left mastoid. His past history was that after a bad fall on his head at the age of 2 yr., his left drum membrane was perforated, and he has had intermittent drainage from the left ear since then. Several weeks before admission he had a complete facial paralysis on the left side which still existed at the time of examination.

An aural polyp was discovered during examination of the left external auditory canal, and after removal of the polyp it was seen that the posterior half of the drum was missing and a purulent discharge was coming from the middle ear. The cholesteatoma could be seen protruding into the middle-ear area as well as extending through the fistula in the postantral area, and although roentgenograms were not needed for the diagnosis of the cholesteatoma they were taken to see the extent of the erosion internally.

A radical mastoidectomy disclosed an erosion of 0.5 cm. which went through the bony tegmen and exposed the dura with a few granulations on its surface. Matter exenterated from the mastoid antrum and middle area was sent to the laboratory and found to be squamous-cell carcinoma. High-voltage x-ray therapy was administered at the Fite Clinic, Muskogee, Okla., by Dr. William Doyle who gave a total dose of 2,000 r of x-rays (200 kv., 8 ma. 50 cm. distance and 0.5 mm. Cu and 1 mm. Al filtration). Since the operation the patient has been observed periodically for six months, with no evident extension of the carcinoma either locally or intracranially, but, of course, this is only the beginning of his observation period.

SUMMARY

A cholesteatoma in the left middle ear of a patient with a history of membrane perforation and intermittent ear drainage since infancy was treated surgically, and laboratory analysis of matter taken from the mastoid antrum and middle area disclosed squamous-cell carcinoma. X-ray therapy was administered, and results after the first six months of observation appear satisfactory.

325 W. Broadway.

† Dr. Coachman died May 24, 1951.

OBLITERATIVE FRONTAL SINUSITIS

Report of a Case and Review of Literature

BENJAMIN M. VOLK, M.D.
AND
S. MUKERJI, M.B.B.S. (Patna, India), D.O.M.S. (London)
ALBANY, N. Y.

OBLITERATIVE frontal sinusitis as a sequela to chronic infection of the frontal sinus is an infrequent occurrence. Tilley [1] was the first who called attention to this form of sinusitis in an address before the Royal Society of Medicine in London in 1928. In 1936, Skillern [2] gave a comprehensive report about the pathological process of the disease and described five such cases in which treatment with radical surgery was successful.

The process of obliteration of the sinus starts with a low-grade-hyperplastic mucosal inflammation of slow development resulting in the formation of granulations and involvement of the periosteum and ultimately in a thickening osteitis. It is an "osteogenesis" in contrast to "osteomyelitis." The latter is a destructive osteitis, the tissues being overwhelmed and destroyed by invading organisms. In osteogenesis the tissues proliferate, walling off or encapsulating the invading organism.

The exact process starts, after an infection, from within the sinus cavity where the periosteum below the lining membrane is so traumatized by the disease that it becomes congested and thickened, assuming the appearance of granulation tissue. The latter serves as a base of an embryonic character where rapid differentiation occurs of some of the cells into osteoblasts. Calcium deposits later on complete the process of ossification. Whether or not the infection leads to osteomyelitis depends on the amount of thickness there is in the compact bone separating the infection from the diploë. If the compact bone is of adequate thickness and if the person's resistance is capable of responding to the bacterial stimulus by an excitation of the osteoblasts, osteogenesis occurs as a protective mechanism to prevent infection invading the diploë and calvarium.

The chief diagnostic features of this disease process are episodes of previous attacks of frontal sinusitis and a roentgenogram showing a central limitation of the growth at the intersinus septum, with thick eburnated bone forming the floor of the sinus, walling it off from the nasal cavity. Examination of a culture grown from specimens from the pockets or recesses in the sinus demonstrates the infective nature of the disease to differentiate it from simple "aplasia" of the frontal sinus or any benign or malignant growths.

From the Department of Otolaryngology, Albany Medical College and Albany Hospital.

1. Tilley, T. L.: Obliterative Frontal Sinusitis, Proc. Roy. Soc. Med. **21**:1574 (May) 1928.
2. Skillern, S. R.: Obliterative Frontal Sinusitis, Arch. Otolaryng. **23**:267 (March) 1936.

REPORT OF A CASE

F. H., a white male engineer, aged 54, was first seen on May 17, 1950, complaining of acute pain in the frontal region following an attack of a head cold two weeks previously. The character of the pain was steady and constant, and there was a swelling of the forehead in the midline about 2 by 3 cm. which was tender on pressure.

According to the past history the patient underwent a nasal polypectomy in 1939. He had frequent attacks of sinusitis, and a ureteric calculus was found in 1946.

The patient was hospitalized on May 19 for an attack of acute suppurative frontal sinusitis. An intensive course of aureomycin and penicillin was administered for 15 days, after which, on June 2, the patient was discharged from the hospital apparently cured. Roentgenograms of the

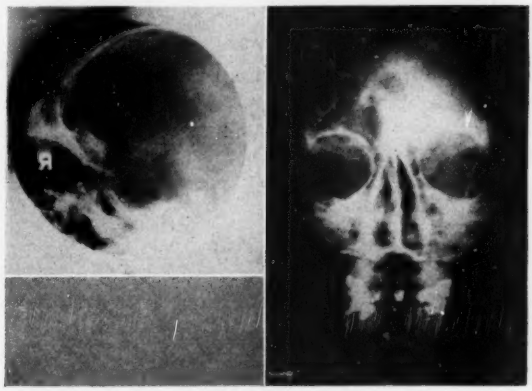


Fig. 1.—Lateral and axiopupal roentgenograms before operation.

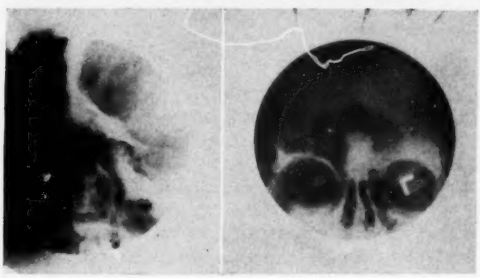


Fig. 2.—Lateral and axiopupal roentgenograms after operation.

sinuses during this period showed an increased density of the left frontal sinus, bony in character. The patient came back to the hospital on July 19, with reappearance of the symptoms, dull pain and a sense of weight over the frontal area, evidence of slight toxemia and recurrence of the midline swelling on the forehead.

The results of systemic examination were normal. Rhinological examination showed only a hypertrophic mucous membrane with no evident discharge. Neurological examination showed nothing significant. Locally, on the forehead, was a fluctuant mass 1.5 cm. above the glabella and about 2.5 cm. in diameter. The roentgenologic report was similar to the previous one—"irregular sclerosis of the left frontal sinus, but no evidence of acute destructive process." An aspiration of the swelling yielded a thick, greenish pus, which on culture revealed Micrococcus tetragenus and diphtheroids. A provisional diagnosis of obliterative frontal sinusitis was made and a radical operation planned.

Under general anesthesia a horizontal incision aabout 15 cm. long was made about 2 cm. above the supraorbital margins and the bony forehead exposed. In the center of the field was found a pocket of eroded bone about 2 cm. in depth containing thick, dirty, green purulent matter. The pocket had no connection with either of the sinuses. Exploration of the right sinus showed a perfectly normal cavity. The left sinus, on exploration, was found to be entirely absent, its place instead being filled with dense eburnated cancellous bone. This new bone was thoroughly chiseled and curetted until the smooth, hard, bony surface of the posterior wall of the sinus was reached. After further curetting, both the sinuses were made into one large cavity. There was no evidence of a left nasofrontal duct, the right one being present normally. The operation was completed and skin and periosteum sutured and two Penrose drains put at the two angles of the wound.

On the ninth day after the operation the patient was discharged with complete relief of all pain and the wound perfectly healed.

SUMMARY

A case of obliterative frontal sinusitis has been reported together with a brief review of the existing literature.

Clinical Notes; New Instruments and Techniques

NASAL PACKING

The Rubber Pneumatic Pack

RALPH W. STEVENS, M.D.
WALLA WALLA, WASH.

An improved model of the rubber pneumatic nasal pack previously described [1] has been developed after 15 yr. of experience with nasal packing methods. The earlier model of the nasal pack (Fig. 1) was unavailable during World War II owing to the rubber shortage, and the present change was felt advisable when production was resumed.

The old model of the pack without the breathing tube was found to have several minor disadvantages. In the case of a very large nasal fossa, the flat pack assumed an ovoid cross section on adequate inflation which allowed the pack to slip through the posterior choana like an olive-tipped bougie. In one case of postoperative polypectomy with considerable turbinate

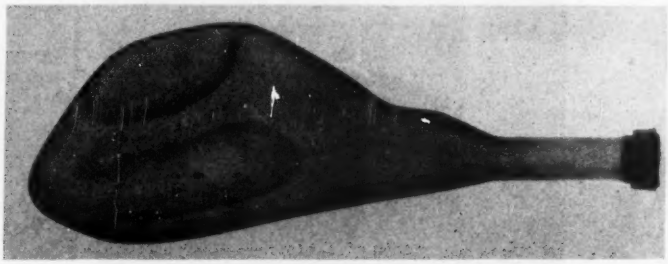


Fig. 1.—Old model nasal pack.

atrophy the inflated pack, fastened to the cheek by adhesive tape, was swallowed by a senile patient, causing considerable apprehension until the pack appeared in the patient's stools within 24 hr. (still inflated). The small vestibular portion of the pack was also found to be inadequate to pack the external vestibule of the nose, requiring cotton-wool packing in addition. Lastly, the rubber cork used for injection or withdrawal of air was rather small and permitted the needle to puncture the thin wall of the pack inadvertently.

The nasal pack with the stiff breathing tube was found harder to introduce, more uncomfortable, and not too effective, since the tube filled with secretions within several hours. Because of these disadvantages, it was felt advisable to discontinue making the pack with the breathing tube until these problems could be solved.

The new model nasal pack [2] (Fig. 2) was designed to remedy the above disadvantages. The shape was made more rectangular, giving more adequate packing of the anterior-superior septal space under the vault of the nose and the posterior choana under the sphenoid sinus. The vestibular portion of the pack was enlarged to fill the vestibule of the nose. This enlarged portion when inflated is anterior to the narrow rima maxillaris and helps prevents the pack from slipping posteriorly. The depressions, or grooves, in the pack designed to fit the turbinates were made deeper, allowing better contact and keeping the pack flatter (less ovoid) when

1. Stevens, R. W.: Improved Intranasal Packing: A Rubber Pneumatic Pack, Arch. Otolaryng. **23**:232 (Feb.) 1936.

2. The pneumatic nasal pack is available from the American Cystoscope Makers, Inc., 1241 Lafayette Ave., New York, and from dealers.

inflated. The self-sealing rubber cork was increased to 5/8 in. (1.55 cm.) in length, with a small hollow core in the distal half of the cork to protect the thin wall of the pack from penetration by the needle, provided a needle no longer than 5/8 in. is used.

The advantages of the pneumatic pack have been significant during the 15 yr. of observation. While the idea was at first thought original, further investigation disclosed that an inflatable bag for controlling hemorrhage was patented in 1849 by Haile (U. S. Patent No. 6,796) and in 1886 by Ashley (U. S. Patent No. 346,633). An inflatable nasal bag with a breathing tube in the center was invented by an Italian, Dionisio Ignazio, in 1889 (Italian Patent No. 1,839).

After the appearance of my article in 1936, correspondence from Dr. August L. Beck of New Rochelle, N. Y., revealed that he had used an inflatable rubber nasal pack since 1916, with excellent results.[3] The Beck nasal pack consisted of a trapezoidal-shaped rubber bag flat on each side and connected to a rubber inlet tube which was tied with string to prevent loss of pressure. The dimensions of the pack were sufficient only to exert lateral

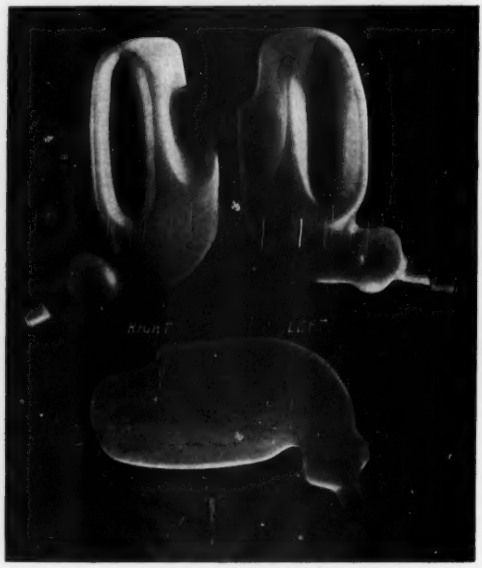


Fig. 2.—New model nasal pack.

pressure on the bony portions of the nasal wall and opposite septum. Difficulties in obtaining adequate production of the Beck pack, rather than any defects inherent in the principle of an inflatable pack, prevented widespread use and familiarity.

Pressure on either side of the nasal fossa, where the greatest area of surface mucosa and vessels occurs, is difficult to obtain and control with other methods of packing. The amount of swelling of hydroscopic splints is unpredictable and uncontrollable. In an open posterior choana there is no firm tissue posteriorly for gauze packing to be packed against. With gauze or cotton packing, it is necessary to jam a sufficient quantity between the turbinates and the septum to give resistance to packing placed anteriorly. The pneumatic pack exerts even pressure in all directions and is very effective laterally where the pressure is most needed.

Since its development, the pneumatic rubber pack has been found very efficient in controlling either postoperative or spontaneous nasal hemorrhage. There have been no untoward results when the packs have been used properly. In no case was a perforation of the septum felt to

3. Beck, A. L.: On Inflatable Rubber Bag for Intranasal Use, Am. J. Surg. **31**:77, 1917.

be the result of use of the packs. When a perforation did occur, it was always associated with opposing unrepaired tears of each mucoperichondrium. There have been no cases of necrosis of the nasal mucosa from excessive pressure. Actual measurement disclosed that the usual pressure amounted to 30 mg. of mercury when 15 cc. of air was used in the pack for the average nasal fossa. The ideal pressure can be easily gauged by the amount of rebound of the syringe plunger when the thumb is released. The rebound should be relatively slow unless a greatly increased pressure is needed temporarily to control hemorrhage. Owing to the unavailability of the packs during the war, one pair was used repeatedly for six years satisfactorily in spite of a tendency of the packs to leak air through the rubber cork after repeated needle punctures. In fact, this leakage was regarded as a beneficial condition, in that the pack automatically decompressed itself as turgescence of the tissue developed postoperatively. As much as 5 to 8 cc. of air was often found to have escaped when the packs were removed. Some air is lost even with the new improved rubber cork, probably diffusing through the thin rubber wall of the pack as it would from a balloon or a tire inner tube. This loss of air undoubtedly makes the patient more comfortable as the postoperative tissue-swelling begins.

The pack has been especially valuable in controlling nasal hemorrhage of undetermined origin. In almost all cases of epistaxis the pack can be slipped behind a badly deflected septum and then inflated. In several cases, severe epistaxis from beneath the posterior third of the inferior turbinate has been controlled without recourse to carotid-artery ligation. Another valuable feature has been the avoidance of postnasal packing. The packs are encouraged to slip posteriorly and then inflated to the desired size, a pack being used in each fossa and the inlet tubes secured carefully so that the packs can slip only as much as needed.

While any type of nasal packing is uncomfortable to the patient, there is less discomfort from the pneumatic pack because the pressure is evenly distributed. The rubber permits drainage of nasal secretions anteriorly and posteriorly and does not adhere to the mucosa upon withdrawal. The nasal lacrimal duct may be blocked, and the patient may be bothered with epiphora, but this probably also results from stimulation of the nasociliary nerves. An important feature to both the patient and the surgeon is the fact that the pressure can be adjusted easily and simply without removing the pack.

DIRECTIONS

1. The packs are contoured to fit each nasal fossa, there being a right-hand and a left-hand pack. The grooves, or depressions, are for apposition to the turbinates, the smooth side being placed toward the septum.

2. Before introduction, the pack is deflated by aspirating the air with a 20-cc. syringe and a 25-gauge 5/8 in. needle, which is inserted into the center of the rubber self-sealing cork until it is felt to penetrate the central hollow core. (Puncture of the thin rubber wall of the pack should be avoided.) The needle and syringe are then withdrawn.

3. The top half of the posterior portion of the collapsed pack is folded down to make it smaller and the folded portion grasped with bayonet forceps.

4. The pack is wetted with sterile water or procaine hydrochloride solution and then introduced into the dilated vestibule and along the nasal floor until only the corked tube remains outside the nose.

5. The apposition on the turbinate side should be re-examined and the folded superior portion of the pack raised to let it slip between the middle turbinate and the septum.

6. If the opposite nasal fossa is to be packed, the same procedure as in Step 5 should be followed before inflation.

7. The packs are then inflated by the injection of 15 to 20 cc. of air (depending on the size of the fossa) into each pack with the same syringe and needle described in Step 2. An estimate of the pressure can be made from the force necessary to prevent the syringe plunger from sliding back when the thumb is released slightly. Caution must be used to avoid too much pressure. Tourniquet precautions should be followed if excessive pressure is needed to control hemorrhage.

8. The packs are held in place by fastening the cork tubes together with a rubber band, a small ball of cotton having been first placed between them to avoid constriction of the

columella. If only one pack is used, the tube can be taped to the cheek or top of the nasal tip to prevent backward displacement.

9. The packs can be left in place 12 to 24 hr., more air being added or withdrawn if necessary without disturbing the pack.

10. To remove the packs, all the air is aspirated through the cork as before, and the collapsed pack is easily withdrawn from the nose and reinflated before the needle is removed.

11. The packs are cleaned with soap and running water, rinsed with 70% alcohol, and dried for further use. It is best to store them at normal inflation in a closed container and to prevent contact between them with several layers of gauze.

12. Sterilization of the packs before use is easily accomplished by autoclaving or by immersion in 70% alcohol or any of the aqueous germicides for five minutes.

SUMMARY

The advantages of the improved model of the pneumatic rubber nasal pack are as follows: 1. The hollow thin-walled latex-rubber pack is contoured to fit the nasal fossa more exactly than was the old nasal pack. 2. The pack, when collapsed to as small a volume as possible, is easily introduced into the nasal passages. 3. Intranasal pressure is controlled by injecting a known quantity of air. 4. Later modification of this pressure can be made without removing the pack. 5. The patient obtains more comfort from equally applied pressure, which can be less than that applied locally by other methods. 6. A rubber breathing tube may be used optionally. 7. The pack is easily removed by first withdrawing all the air. 8. A rubber self-sealing or protected injection cork acts as an automatic valve. 9. The pack may be used time and again.

308 Baker Bldg.

NEW TECHNIQUE IN THE USE OF A SURGICAL TRAY

JOSEPH JEROME LITTELL

INDIANAPOLIS

During my surgical experience it has seemed most desirable to develop an arrangement whereby instruments would be more quickly and easily available to the field of operation. The previous attempt nearest to this is the Mayo table which, of course, is a stand with sufficient

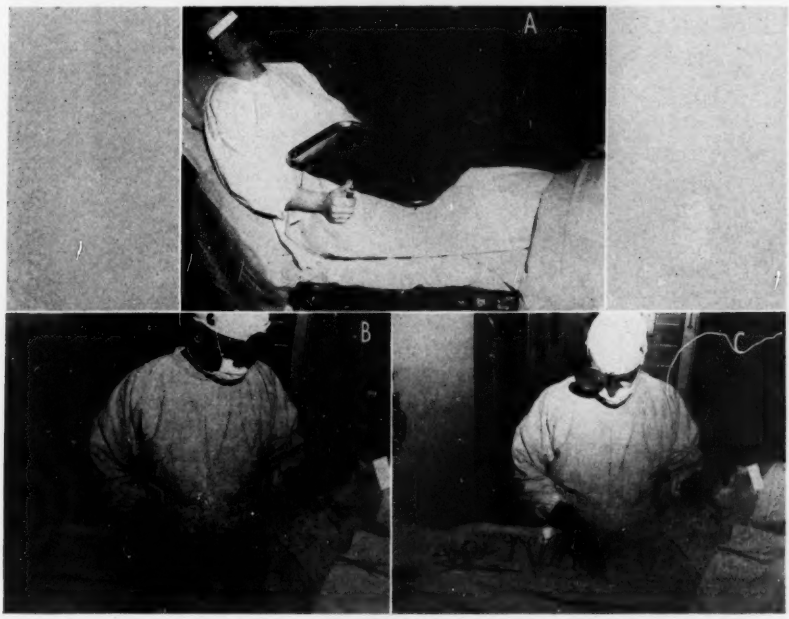


Fig. 1.—*A*, the tray is supported on the patient's thighs below and on the abdomen or chest above. The tray is made level by the adjustment upward or downward of the upper half or the lower half of the surgical table. The clamps which fasten onto the sides are in this case ordinary large storage battery clamps. An improved and more finished product is being developed. *B*, the tray covered with instruments is shown, in this case for a tonsillectomy. I now prefer to place a towel on top of the sheet instead of beneath it as formerly. The instruments may then be moved en masse. *C*, the small arc in which the head moves to choose the proper instrument is indicated.

floor support extending beneath the operating table to hold a rack upon which fits a tray. This tray then, properly and separately draped, extends over the operating table and some inches above the patient.

I have found myself placing instruments, between periods of use, upon the patient's abdomen or chest in an area closer to the operating field than is allowed by the Mayo table. The defects of this procedure are, of course, apparent in that the instruments roll off or become displaced.

From the Department of Otolaryngology, Indiana University School of Medicine.

The same need and some degree of solution for it have been evident to others. At the Foman Clinic a spiral spring is pinned across the upper abdomen of the patient and a few instruments are placed in the coils.

A technique is presented here which, I believe, will be found useful in operations on the nose, throat, neck, and upper chest under local anesthesia only. It may be recommended on several counts.

1. The idea is simple. As may be seen in the photographs, a Mayo tray is supported on the patient's abdomen. Clamps which fasten to the tray are grasped in the patient's hands.[1]

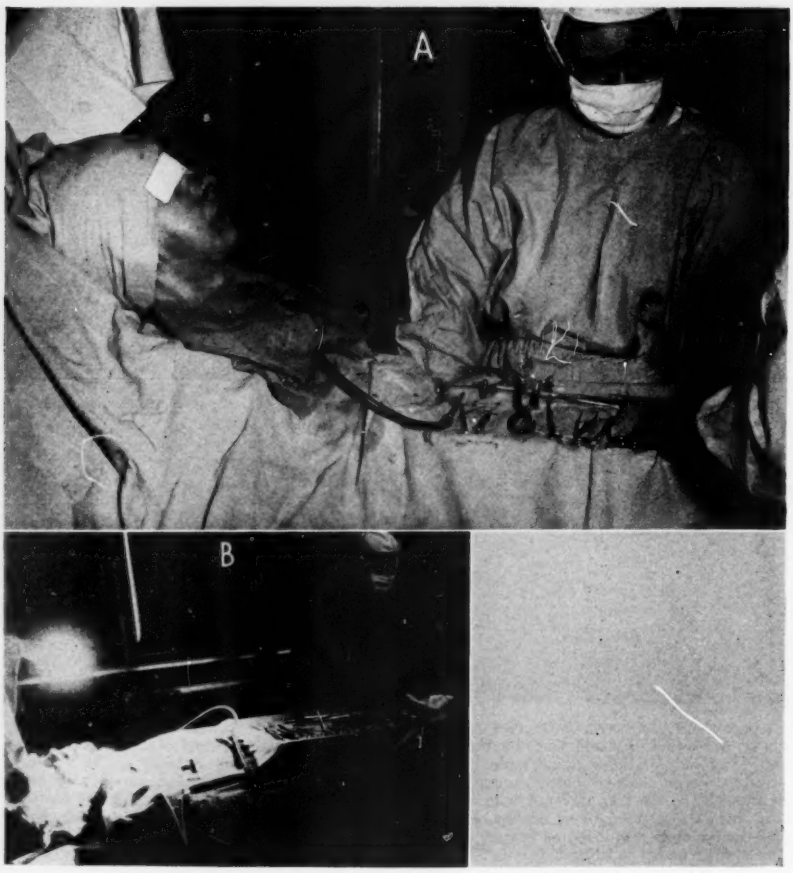


Fig. 2.—*A*, the ready availability of the instruments to the operative field and to the assistant is apparent. The support required by the Mayo table is absent. *B*, use of the tray with the patient in the supine position through adjustment of the levels of the surgical table. In the background is shown a Mayo table, which may be used if desired for solutions or supplies.

2. The tray is supported with its long dimension parallel to the axis of the body; therefore, the tray does not obtrude itself into the field of movements of the operator. There is no support, such as is present in the Mayo stand, to separate the assistant from the surgical field.

1. Manufactured by Emmerich Schmid, Inc., 1501 Madison Ave., Indianapolis.

3. All the instruments can be kept within easy view and easy reach of the operator; his eye and hand need to travel only a very small arc between the point of surgical interest and the instrument which is applicable to that instant's surgical need. The surgical field and the instruments are approximately the same distance from the eye. Therefore, no change of accommodation is necessary. This is of great value to the presbyopic surgeon. For the above reasons, less time and energy are consumed. This is of value to surgeon and patient alike.

4. The patient finds the tray comfortable to support and holding onto the handles reassuring. During my period of observation, the patient has been quieter because of his feeling of responsibility and co-operation, and at no time has the tray been more than slightly moved.

5. Surgical nurses highly approve this technique as there is one less table to drape. The sterile sheet which routinely covers the patient covers the tray also. A sterile towel is placed over the area which the tray occupies.

CONCLUSIONS

I am presenting a technique in which a tray rests below on the thighs of the patient, and above on the upper abdomen. The tray is covered with the same sheet which covers the patient routinely. The instruments are readily and quickly available. The assistant has a more ready access to the surgical field. The end result is a more efficient, less fatiguing experience for both the surgeon and the patient, as well as less trouble for the nursing staff.

603 Hume Mansur Building (4).

TRACTION SUTURES IN TONSILLECTOMY

RAFAEL C. TIONLOC, M.D.
MANILA, PHILIPPINES

Advances in laryngology have been made and are permanent. Just as appendectomy is the first step for an abdominal surgeon, so tonsillectomy is the first rung on the ladder of success for a laryngologist. As these operations are performed, the surgeons always try to improve the technique and make the procedure easy for both the patient and the surgeon.

The method of removing tonsils is either partial removal (tonsillotomy) or complete removal (tonsillectomy). The complete removal is an old way in surgery and was practiced by Celsus [1] before the Christian era (53 B. C. to 7 A. D.). The operation can be done by a rapid, or guillotine, method or by slow dissection. Nowadays, the majority of laryngologists, following the experts of England and America, agree that if a tonsil is diseased, surgical intervention is in order. It should be removed complete with its capsule. George E. Waugh of England, according to Thomson and Negus,[1] in 1909 drew attention to the unsatisfactory work done by guillotine, and recommended enucleation by dissection. In the guillotine method some use vulsellum or tenaculum forceps for dragging the tonsil to the median line of the pharynx within the grasp of the tonsillotome. Whillis and Pybus, according to Thomson and Negus,[1] obtained the same results by using the forefinger to dislocate the tonsil from its socket and push it into the ring, or fenestra, of the tonsillotome. Sluder, cited by Thomson and Negus,[1] suggested a point in the lower jaw to push it inward, i. e., the alveolar eminence of the mandible which is just above the mylohyoid line and is produced by the prominence of the last molar tooth in its socket, which is more marked in the young.

For dissection, the use of forceps to grasp the tonsil is unavoidable. Some laryngologists are trained to change the position of the grasp as the dissection of the tonsil reaches the lower pole, while others maintain one position in the grasp of the tonsil during the dissection. The use of traction sutures replacing the forceps in this step of the operation comes into play.

TRACTION SUTURE TECHNIQUE

The following types of traction sutures are used: (1) single horizontal traction suture, (2) single vertical traction suture, (3) double horizontal traction sutures, (4) double vertical traction sutures, and (5) mixed vertical—horizontal traction sutures.

The procedure is the same as that of any tonsillectomy except for the grasping of the tonsil. One or two 0000 nonabsorbable surgical suture threads with full curved atraumatic needles are prepared with needle holders.

1. With the single horizontal traction suture, the needle pierces the tonsil posteriorly at its center, the point of the needle coming out anteriorly. During the procedure the tonsil is not held by forceps. Procautions should be taken to avoid the inclusion pillars.

2. With the single vertical sutures, the needle is made to pierce the center of the tonsil either from the superior or from the inferior pole, according to choice. The point of the needle comes out at the inferior or superior pole. Puncture of the uvula and the tongue should be avoided.

3. The same procedure is followed if the double horizontal, double vertical, and the mixed horizontal-vertical sutures are used, but the sutures are made at an equal distance to the first puncture in either pole of the tonsil.

From the Department of Otolaryngology of the University of Santo Tomas College of Medicine and Surgery.

1. Thomson, St. C., and Negus, V. E.: Diseases of the Nose and Throat: A Textbook for Students and Practitioners, ed. 5, London, Cassell & Co., Ltd., 1948, pp. 429-452.

After insertion of the suture, slight traction will determine whether or not the tonsil substance is strong enough to resist the pull. In this way the amount of traction allowed without danger of rupture can be calculated. Traction is created by pulling the ends of the thread in such a way as to bring the tonsil toward the midline of the pharynx. Then the incision of the mucous membrane between the pillars and the tonsillar substance, either anteriorly or posteriorly, is made with a knife or a blunt tonsil dissector. Then the incision is widened by a tonsil dissector; stroking from within outward will separate the mucous membrane and loosen the upper pole of the tonsil from the anterior and posterior piflars. The upper pole is then separated from its bed, and dissection is carried downward to the lower pole. The tonsil then hangs freely at its pedicle and is held by the thread. The snare is slipped easily to the ends of the thread and passed down to the lower pole where the snaring is accomplished. The tonsil is thereby completely severed and removed with the thread. Routine hemostasia is performed.

ADVANTAGE OF TRACTION SUTURES OVER VULSELLUM OR TENACULUM FORCEPS

1. The traction produced by the nonabsorbable surgical suture exerts little and equal pressure on the tonsil, and hence tonsil destruction, which usually occurs with forceps, is avoided.

2. Because rotation or traction of the tonsil is easier in all directions and the traction is equal the pillars are more prominent and tense for recognition and incision of the plicae.

3. It is easy to slip the snare to the ends of the thread.

4. The suture serves to hold the tonsil and prevents it from falling to the esophagus or hypopharynx during the process of snaring, especially when the gullotine method is used.

5. There is a wider surgical field, especially when children and patients with small mouths are operated on.

6. In sensitive patients, gagging is minimized.

7. The patient can easily spit out the excess blood in his mouth without the surgeon removing the traction suture, whereas forceps must be removed every time the patient spits.

8. The procedure is simple and practical, of more value with friable and submerged varieties of tonsils, and a very easy method for the removal of ordinary soft and scirrhous tonsils.

Surprisingly, bleeding of the tonsil substance is less than expected during the insertion of the traction sutures. The use of traction sutures in tonsillectomy is not proposed to change the operative technique or to dispense with the forceps but as a suggestion for trial.

OTOSCLEROSIS AND ACTH

Preliminary Report

ZHIVKO ANGELUSCHEFF, M.D.
NEW YORK

In March, 1950, I presented a review of the laboratory and clinical results obtained with corticotrophin and cortisone to the otolaryngological service of the New York City Hospital. On the basis of these findings speculations were made as to the possibility of inhibiting osteogenesis during the fenestration operation.

The fenestration operation for clinical otosclerosis has often been accompanied with many disappointments. The natural forces which tend toward osteogenetic repair have stimulated the efforts of physicians in various directions in an attempt to overcome this great barrier to complete success. I believe that since all technical efforts have in the long run been ineffective in inhibiting osteogenesis, a new biological orientation should be sought.

The relatively short experience with corticotrophin and cortisone has already furnished rather impressive evidence as to their effect on osteogenetic metabolism. The inhibiting of osteogenetic and mesenchymal elements by corticotrophin without interference with epithelial proliferation may be exceedingly significant in maintenance of the patent fistula. The rationalization for its use may be its depression of the repair elements such as granulation tissue, histiocytes, fibroblasts, new blood vessels, and calluses.

In the light of the fact that a new biological approach to the problem of maintenance of the patent fistula in fenestration is being sought, I wish to present this preliminary report in the hope that it will stimulate investigation with adequate control by those clinicians all over the country who are now actively engaged in fenestration surgery for the relief of clinical otosclerosis.

120 East Eighty-Sixth Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTIONAL EXAMINATION OF HEARING

ALFRED LEWY, M.D.
SHERMAN L. SHAPIRO, M.D.
AND
NORMAN LESHIN, M.D.
CHICAGO

CONTRIBUTIONS to the physiology and psychology of hearing and articles dealing with acoustic physics are apparently on the increase, and, as in the previous year, much credit goes to the Scandinavian countries, with the United States not far behind. On the other hand, there is an apparent decrease in contributions on the functional tests of hearing of immediate interest to the clinician. Rehabilitation continues its upward trend, and the number, and we hope the quality, of hearing aids is increasing.

HEARING AIDS

A review of some experiments with binaural hearing aids is reported by Hirsh.[1] The binaural summation averaged 3 db. for pure tones and the same for speech intelligibility at threshold. Smaller differences in frequency also can be detected binaurally. In the presence of a masking noise this summation decreases or disappears. At 35 db. there is a summation of loudness of 6 db. Using a monaural hearing aid in noisy surroundings may be disagreeable to the user. It is impossible to localize separately desired and undesired sources of sound, but it is possible with separate microphones preferably mounted on each ear. One microphone and two earpieces have no advantage over monaural aids.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

As of Aug. 1, 1951

Audicon Models 400 and 415
Mfr., National Earphone Co., Inc.
20-22 Shipman St.
Newark 2, N. J.

Audivox Model Super 67
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
259 W. 14th St.
New York 11

Aurex Model F
Aurex Model H
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Harmony Mono-Pac
Beltone Symphonette Model

1. Hirsh, I. J.: Binaural Hearing Aids: Review of Some Experiments, J. Speech & Hearing Disorders **15**:114, 1950.

Beltone Mono-Pac Model M
Mfr., Beltone Hearing Aid Company
1450 W. 19th St.
Chicago 8

Cleartone Model 500

Cleartone Regency Model
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1
Mfr., The Dahlberg Company
2730 W. Lake St.
Minneapolis 16

Dysonic Model 1
Mfr., Dynamic Hearing Aids, Inc.
1042 Atlantic Ave.
Brooklyn 16

Electroear Model C
Mfr., American Earphone Co., Inc.
10 E. 43rd St.
New York 17

Gem Model V-35

Gem Model V-60
Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Maico Atomeer

Maico UE Atomeer

Maico Quiet Ear Models G and H

Maico Model J
Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Mears (Crystal and Magnetic) Aurophone Model 200

1947-Mears Aurophone Model 98
Mfr., Mears Radio Hearing Device Corp.
1 W. 34th St.
New York 1

Micronic (Magnetic Receiver) Model 101

Micronic Model 303

Micronic Star Model
Mfr., Micronic Company
727 Atlantic Ave.
Boston 11
(See Silver Micronic)

Microtone Audiomatic T-3

Microtone Audiomatic T-5

Microtone Classic Model T-9

Microtone Model T-10

Microtone Model 45
Mfr., The Microtone Company
Ford Parkway on the Mississippi
St. Paul 1

National Cub Model (C)

National Standard Model (T)

National Star Model (S)

National Ultrathin Model 504

National Vanity Model 506
Mfr., National Hearing Aid Laboratories
815 S. Hill St.
Los Angeles 14

Otarion Model E-1S

Otarion Model E-2

Otarion Model E-4

Otarion Models F-1, F-2, and F-3

Otarion Model G-1 (Whisperwate)
Mfr., Otarion, Inc.
159 N. Dearborn St.
Chicago 1

Paravox Model J (Tiny-Myte)

Paravox Models VH and VL (Standard)

Paravox Model XT ('Xtra-Thin)

Paravox Model XTS ('Xtra-Thin)

Paravox Model Y (YM, YC, and YC-7) (Veri-Small)
Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear Permo-Magnetic (Multipower)

Radioear Permo-Magnetic (Uniphone)

Radioear All-Magnetic Model 55

Radioear Model 62 Starlet

Radioear Model 72

Mfr., E. A. Myers & Sons
306-308 Beverly Rd.
Mt. Lebanon
Pittsburgh 16

Rochester Model R-1

Rochester Model R-2

Mfr., Rochester Acoustical Laboratories Inc.,
117 Fourth St., S.W. Rochester, Minn.

Silver Micronic (Magnetic and Crystal) Models 202M and 202C

Mfr., Micronic Company
727 Atlantic Ave.
Boston 11
(See Micronic)

Silvertone Model 103BM

Mfr., National Hearing Aid Laboratories
815 S. Hill St.
Los Angeles 14
Distr., Sears, Roebuck & Company
925 S. Homan Ave.
Chicago 7

Silvertone Model J-92

Mfr., Sears, Roebuck & Company
925 S. Homan Ave.
Chicago 7

Silvertone Model M-35

Mfr., Micronic Company
727 Atlantic Ave.
Boston 11
Distr., Sears, Roebuck & Company
925 S. Homan Ave.
Chicago 7

Silvertone Model P-15

Mfr., W. E. Johnston Mfg. Co.
708 W. 40th St.
Minneapolis 8
Distr., Sears, Roebuck & Company
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99

Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Model 600

Sonotone Model 700

Sonotone Model 900

Sonotone Models 910 and 920

Sonotone Model 925

Sonotone Model 940

Mfr., Sonotone Corporation
Elmsford, N. Y.

Super-Fonic Hearing Aid.

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Televox Model E

Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Model 22

Telex Model 97

Telex Model 99

Telex Model 200

Telex Model 300-B

Telex Model 400

Telex Model 1700

Mfr., Telex, Inc.
Telex Park
Minneapolis 1

Tonamic Model 50

Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Model Royal

Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Trimm Vacuum Tube Model 300

Mfr., Trimm, Inc.
400 Lake St.
(P. O. Box 489)
Libertyville, Ill.

Unex Model A

Unex Midget Model 95

Unex Midget Model 110

Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Model J
Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Western Electric Models 65 and 66
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
259 W. 14th St.
New York 11

Zenith Model 75

Zenith Miniature 75

Zenith Model Royal
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Aurex (Semi-Portable)
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid
Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr., Sonotone Corporation
Elmsford, N. Y.

HEARING TESTS

Acuity and perceptive accuracy of monaural and binaural hearing was investigated by Poulas.[2] A pure-tone audiometer was first used to test for acuity. A record-player and phone attachment was set up. The intensity level of the sentences was decreased by 2-db. steps. The subjects were elementary or high-school pupils. Two hundred and twenty-eight tests were administered. The scores of 147 were better for binaural hearing. Thirty-four had lower scores for binaural than for monaural hearing; 47 had the same score for both.

On the percentage basis, 63.6% showed better scores for binaural hearing; 20.6% had the same score for both; 15.8% showed decreased binaural hearing compared with monaural.

Leisti,[3] of Turku, Finland, presents report on a series of 451 patients, ranging in age from 16 to 80 yr. He credits Bunch as the first to make investigations of presbyacusis with an audiometer, and he also cites Ciocco's findings. He finds that the auditory acuity decreases continually with advancing age, and the decrease becomes noticeable as early as between 20 and 30 yr. The deterioration is very slow and affects mainly the high-tone range from c^5 upward. Below this limit the hearing loss is insignificant up to the age of 50. After 50 the impairment is more apparent from c^2 and c^3 upward and appears to be greater in men than in women. He believes this difference to be due to external causes and admits that it is not statistically conclusive. The results are shown in five tables and 12 diagrams.

2. Poulas, T. H.: Acuity and Perceptive Accuracy of Monaural and Binaural Hearing, Volta Review **52**:314 (July) 1950.

3. Leisti, T. J.: Audiometric Studies of Presbyacusis, Acta oto-laryng. **37**:555 (Dec.) 1949.

Edward B. Newman, of Harvard University, reviews the progress made during the past year in the field of hearing.[4] In detail his review is principally of interest to acoustic physicists and physiologists, but practicing otologists may glean some valuable information therefrom.

The technical improvements, such as the systematic use of noise and of recorded speech as a stimulus, have led to greater precision in the exploration of old problems. Experimenters have had to become more expert in related fields, such as the physics of sound and mathematics.

Physiological work is still handicapped by lack of detailed knowledge of anatomy. The basic theoretical issues of hearing are still unsolved. On the one hand, the resonance theory and the telephone theory are almost certainly wrong in detail. There is neither a tuned string nor a linear transducer in the cochlea, but each of these older theories, if stated in most general terms, is partly correct. The author feels sure there is some degree of mechanical analysis in the inner ear, but the general formulations do not help much in finding the correct details. The same may be said for the theories about the central nervous system. The nervous system is an exceedingly intricate mechanism. What is needed is not more speculation but more powerful experimental tools.

An article on the streptomycin treatment of laryngeal tuberculosis [5] again calls attention to the phenomenon of shortened bone conduction, with retention of good hearing by air conduction, occurring in tuberculous patients. [Many years ago O. Beck called attention to this same phenomenon in syphilis.—*Abstracter*]

A rather detailed examination of the residual hearing capacity of pupils in schools for the deaf was made by Goodman.[6] An effort was also made to obtain data for the correlation of hearing capacity with the causation of the deafness. Three hundred and forty-three pupils between the ages of 8 and 12 yr. were tested, 243 in residential and 100 in day schools for the deaf.

Meningitis more commonly caused severe deafness than otitis media; congenital conditions were intermediate between the two. Otitis media was surprisingly common in children in whom it was not the primary cause of the deafness. In the vast majority the deafness dated from before 5 yr. of age. One child with good hearing had aphasia (anomia). A large proportion of children who had useful residual hearing for speech were deaf from birth.

No close correlation could be found between the percentage loss of useful hearing, as estimated from the pure-tone audiograms by the Sabine-Fowler method, and the scores on tests with the speech audiometer. The teachers from experience in the classes could estimate extremely well their pupils' ability to hear speech.

In a paper read before the Royal Society of Medicine, Section of Otology, Dix and co-authors [7] presented a refinement in the differential diagnosis of so-called nerve-deafness. The tuning-fork tests make it possible to effect a reasonably

4. Newman, E. B.: Hearing, Ann. Rev. Psychol. **1**:49, 1950.

5. Germán, T., and Nákó, A.: Observations on the Streptomycin Treatment of Laryngeal Tuberculosis: Some Aspects on the Streptomycin-Vestibular Damages, Acta oto-laryng. **38**:97 (April) 1950.

6. Goodman, A. I.: Residual Capacity to Hear of Pupils in Schools for the Deaf, J. Laryng. & Otol. **63**:551 (Oct.) 1949.

7. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: "Nerve" Deafness: Its Clinical Criteria, Old and New, J. Laryng. & Otol. **63**:685 (Nov.) 1949.

clear-cut distinction between perception deafness and deafness due to disturbance of the sound-conducting apparatus of the middle ear, but these tests do not make it possible to say what the particular components of the perceptive mechanism are and in which way they have been affected. In the last 10 years more information has been available concerning the histopathology of the inner ear and the eighth nerve, and new knowledge of cochlear function has led to new test procedures. These new tests give promise of achievement of some insight into the nature and localization within the perception mechanism of the pathological process concerned.

The new procedures are (1) the loudness-recruitment test and (2) the intelligibility test for amplified speech. These tests have been systematically applied in two groups of subjects, those with Ménière's disease and those with eighth-nerve neurofibroma. In Ménière's disease it is believed that the disturbance of the cochlear function is due to a disorder of Corti's organ. In neurofibroma it is known there occurs a degeneration of the cochlear-nerve fibers, with preservation of Corti's organ. There is this to be considered: If the pressure of the tumor or section of the nerve interferes with the blood supply, then various degrees of necrosis are to be seen in any or all of the cochlear structures. In Ménière's disease the primary change within the labyrinth is distention of the endolymph system. Briefly, the authors claim that in Ménière's disease there occurs a primary disorder of the hair cells of Corti's organ, with preservation of the cochlear-nerve fibers, while in eighth-nerve neurofibroma the cochlear-nerve fibers are affected, the hair cells being preserved.

In middle-ear deafness there is no recruitment. For instance, if there is a threshold loss of 30 db. at 1000 cps as compared with the other ear, the difference in subjective sound remains the same at any intensity. On the other hand, in nerve-deafness the sensitivity loss becomes progressively less as the intensity increases, and at, say, 80 db. equal intensity evokes equal loudness response. In eighth-nerve neurofibroma, however, recruitment is characteristically not present at all. The type of response resembles that of conduction deafness. If an incomplete recruitment is found, the authors attribute it to involvement of the cochlear blood supply by tumor pressure.

The use of the intelligibility test for amplified speech is explained as follows: In nerve-deafness inability to understand speech is out of proportion to the threshold loss as shown by the audiometer. With conductive deafness this disproportion between hearing and understanding is not present. If the sounds reach the cochlea, e. g., by means of a hearing aid, intelligibility is restored. In nerve-deafness restoration of intelligibility for speech by mere amplification is less successful.

In Ménière's disease recruitment of loudness is the rule, but recruitment of intelligibility may be conspicuously absent. It may even occur that as loudness increases intelligibility falls off. In neurofibroma intelligibility increases with loudness.

These tests also explain the diagnostic error of mistaking an early neurofibroma for an early unilateral otosclerosis. The article is well illustrated by diagrams.

Dr. S. R. Silverman, director of the Central Institute for the Deaf, St. Louis, presented an article [8] before the British Royal Society of Medicine in which he

8. Silverman, S. R.: Recent Developments in Auditory Tests, J. Laryng. & Otol. **64**:23 (Jan.) 1950.

discussed the principles underlying the articulation function, which expresses the relation between words correctly heard and intensity. He stated that progress has been made in the last decade in the development of articulation tests for clinical purposes. The articulation test as a measure of hearing and discrimination was described. Tests for tolerance of loud speech and the ways the articulation test may assist in diagnosis and prognosis were discussed. He also brought out the use of the social adequacy index for evaluation of remedial procedures.

He concluded that articulation tests serve a useful purpose for the clinical otologist. Comments were made concerning the necessary equipment for these tests.

In the discussion that followed, T. G. Wilson contended that use of tuning forks was a better way of making the clinical diagnosis, while tests with the audiometer were better for checking progress or regression in hearing. Hallpike had shown that apparently increased bone conduction under ordinary room conditions was found to be normal or subnormal in the soundproof room. The hearing of the examiner was diminished by the background noise, while that of the patient was not, as he did not hear it. In some cases bone conduction had been improved by fenestration. Wilson cited Lindsay, who said that the greatest pitfall in the measurement of bone conduction in otosclerosis was in masking. Wilson thought the loudness-recruitment phenomenon might prove of help in locating the lesion. Intelligence, as well as intelligibility, must be considered. Dr. D. B. Fry preferred one-syllable words to spondees. Dr. Silverman thought that sometimes sentence tests were better than word tests.

Comparisons between hearing and speech tests during and after exposure to noise were made by Kley.[9] Pure-tone and speech hearing was tested in a number of persons before, during, and after exposure to the Bárány noise apparatus 5 to 30 min. Hearing for pure tones was only slightly impaired (average, 10%) and mostly for c^4 and c^5, but hearing for speech, especially whispered voice, was reduced 1 to 8 m. The hearing loss for whispered voice cannot be explained solely by the loss for pure tones in the high-frequency register. Further arguments for the discrepancy between pure-tone hearing and speech hearing were found in the observations of Mayer, who noted definite improvement in the whispered voice, without improvement in pure-tone hearing, after tapping of the cisterna magna. Kley expressed the belief that the sound-perception apparatus of the inner ear possesses a special function for the integration of speech and assumes that both functions are located in the organ of Corti. [I do not understand how the author failed to attribute the speech-integration function to the higher centers.—*Abstracter*]

An investigation of the masking effect in conduction and in perception deafness was carried out by de Maré and Rösler.[10] When the intensity of the masking noise is the same in relation to the level at the threshold of hearing, the effect is the same for persons with conduction deafness as for persons with normal hearing. If the intensity of the masking noise is increased, it has relatively less effect on

9. Kley, W.: Comparative Studies on Tone and Speech Perception During and After Noise, Ztschr. Laryng. Rhin. Otol. **29**:1 (Jan.) 1950.

10. de Maré, G., and Rösler, G.: Studies on Masking Effect in Conduction and Internal-Ear Deafness, Acta oto-laryng. **38**:179 (April) 1950.

persons with conductive deafness than on persons with normal hearing, resulting in a relative improvement in speech intelligibility (low tone-masking noise). In some cases an objective superiority appears in the person with conductive deafness over the normal person as regards speech intelligibility and the perception of certain sounds [authors' version of paracusia Willisii—*Abstracter*]. In nerve-deaf persons the masking effect is the same as for normal persons or even greater, an observation which may be attributed in part to auditory fatigue.

If corresponding masking curves are produced in given noisy surroundings, an analogous result in persons with normal hearing and in those with impaired hearing in these surroundings is obtained which shows appreciable differences from the results of the investigations carried out in quiet surroundings. The findings were corroborated by the subjective impressions of 300 patients with impaired hearing.

A critical study of theories of and experiments in sound conduction and sound analysis in the ear is undertaken by Kostelijk.[11] After reviewing the resonance hypothesis and the localization hypothesis, the author favors the place theory of frequency reception. He writes that too many anatomicophysical arguments have been advanced against the resonance hypothesis. The localized pitch perception in the cochlea takes into account the hydrodynamic behavior of the inner-ear system. The problem has been approached from an experimental (von Békésy) as well as from a theoretical point of view (Ranke; Reboul; Zwislocki). If the inner ear is treated as a hydrodynamic system contained in a vessel with partly elastic walls, it may be computed that traveling waves sweep along the basilar membrane, giving rise to a local-frequency-dependent vibration maximum. Since the sound-conduction apparatus may be regarded as a complicated mechanical system, this system may be looked upon as a special form of sound receiver. Its physical properties, such as impedance, and the pressure-transformer action of the middle ear are discussed, taking the physical characteristics of sound receivers in general as a starting point.

Fowler [12] gives a complete and thorough discussion of the recruitment phenomenon and presents the newer tests and additional uses for this phenomenon. In cases of impedance or conduction deafness or in the normal ear, an increase in intensity of sound above threshold will be sensed with an orderly logarithmic increase in its loudness. The loudness function in each ear has a fixed value. In an ear having a neural, or "nerve," deafness, the difference in sound above threshold between it and a normal ear at threshold will disappear as sound is increased over threshold, and at high intensities the sound may seem the same in both ears. This phenomenon is known as recruitment of loudness and is pathognomonic of auditory neural lesions. It does not occur in conduction lesions. The test can be done by using twin receivers and switching the sound from one ear to the other. This is called the alternate binaural loudness test. Von Békésy has constructed an audiometer which automatically records the recruitment phenomenon. Several

11. Kostelijk, P. J.: Theories of Hearing: A Critical Study of Theories and Experiments on Sound-Conduction and Sound-Analysis in the Ear, Leyden, University Press, 1950.

12. Fowler, E. P.: The Recruitment of Loudness Phenomenon, Laryngoscope **60**:680 (July) 1950.

variations of the test are mentioned, as by modulating the amplitude of a tone 40 db. over threshold without interrupting it but varying the loudness every half second, and by utilizing the auditory-fatigue phenomenon, in which the threshold of hearing is definitely raised by so-called auditory fatigue. A threshold shift of about 12 db. is considered normal. A threshold shift over 12 db. may be considered an evidence of recruitment.

No recruitment occurs in a totally deafened ear. It cannot be elicited by the binaural loudness technique if both ears have similar neural lesions. One should avoid masking when testing for recruitment. Some block lesions of the ear may present recruitment. Recruitment is greatest immediately above threshold and diminishes in increment as loudness increases. This phenomenon may persist even after equal binaural loudness volumes have been reached. Impedance lesions limit recruitment, and the two ears in spite of recruitment of loudness because of neural lesions will always show some difference in their loudness-balance levels by the exact number of decibels. This is the measure of their difference in impedance, and one can in this way measure the amount of the impedance lesion and the neural lesion with a single test. Recruitment for speech is indicated when in moderately deafened ears (over-all losses of 20 to 35 db.), a 5- to 10-db. increase over threshold in loudness of speech will produce a marked increase in loudness and intelligibility, but an increase of 20 db. or more will cause distortion and confuse the patient. This will not occur in obstructive deafness. When recruitment is absent in the presence of markedly diminished bone conduction, it indicates an impedance lesion probably in the oval- or round-window regions. Recruitment is valuable as a test for malingering. If recruitment is present, the patient is not malingering so far as his neural deafness is concerned. This test is important in cases of otosclerosis with lowered bone conduction; with its use one can differentiate impedance from neural lesion.

Lempert [13] presents his views and the tests he employs for testing pure physiological hearing function. By true hearing function he means the actual hearing of a deafened ear and not the ability of a deafened person to interpret correctly what little he hears. The examination should disclose what each ear hears functionally, why it does not hear normally, whether it can be made to hear with amplified sound and, if so, whether it is in good enough condition to lend itself to medical or surgical therapy which could replace the amplified sound, and, finally, whether the hearing loss is of a progressive nature. The procedure Lempert employs is essentially a very careful history, audiometry for pure-tone sound conducted by a well-trained assistant, tuning-fork test for pure-tone sound, and speech testing for the ability to hear spoken words. Tuning-fork tests are employed to determine in which of the two ears the hearing impairment for air-borne sound is worse, and, secondly, to determine the approximate degree of unused good cochlear-nerve function reserve still existing in an air-conduction-deafened ear which could be utilized advantageously by a surgically reconstructed and functionally improved air-conduction mechanism. For the first purpose the Weber test is employed, using a 512 d. v. steel tuning fork. Several observations are made from the test. The fork is

13. Lempert, J.: Practical Testing for Pure Physiological Hearing Function, Laryngoscope **60**:344 (April) 1950.

heard equally in the two ears when cochlear function is equally good and air conduction is equally good or bad in the two ears. It is not heard equally in the two ears when cochlear function is equally bad, even though air-borne sound is equal or unequal in the two ears. It is heard only in that ear having good cochlear function with much diminished or no cochlear function in the other ear. This holds true whether air-conduction hearing is equal or unequal in the two ears. Finally, it is heard better in the ear with poorer hearing for air-borne sound when cochlear-nerve function is equal in the two ears. To determine unused cochlear reserve, Lempert employs the Rinne test, using the 512, 1024, and 2048 d. v. steel tuning forks, masking the untested ear for both air- and bone-testing. The end point for air-conduction hearing is first determined, and then without further stimulating the fork it is placed over the mastoid and the added hearing timed. To prevent inaccurate and psychologic factors entering into the determinations, he has the patient face him with eyes open and carefully observes the facial expressions and movements of the eyes and head and the type of response with the hand which is used for the signal. Lempert feels that he is able to determine the degree to which the ability of an ear to hear spoken voice has been reduced or improved by carefully taken audiometric readings of both air- and bone-conducted pure-tone sounds. He feels, however, that in mixed forms of deafness speech testing can be a determining factor and govern the advice to be given the patient. When such tests are done, they should be given in a language completely unknown to the patient or with the employment of meaningless words. A list of such words is given. To determine the degree such hearing can be improved while employing such a test, the improvement of hearing obtained with the use of an old-fashioned ear trumpet will represent the minimum amount of improvement that can be expected after the fenestration operation.

Reger[14] discusses the many factors which influence the accuracy of clinical audiometric measurements. Some of these are the differences in tone-presentation techniques used by various audiometrists and the difficulties encountered in evaluating the patient's responses. Variations in test results among different audiometrists will be minimized by the general adoption of a uniform tone-presentation technique. Reger suggests the use of pulse-tone audiometry, which was first employed by him in 1944. The instrument consists essentially of a motor-driven tone-interruption device which can be attached to a commercial audiometer. The purpose of this instrument is to introduce one of four stimulus patterns as one, two, three, or four dashes or tone pulses coincident with the presentation of an attention light. The first pulse pattern may be presented 10 or 5 db. above the approximate threshold, and each succeeding pattern is attenuated 5 db. until either an incorrect or no response is obtained. Each pattern is presented in a similar manner. The lowest intensity level at which at least two of three patterns are recognized correctly is taken as the correct clinical threshold value. A light goes on to alert the patient each time a pulse pattern is introduced. The successive pulse patterns are presented in random order. Pulse-tone audiometry introduces a testing procedure which enables retesting a given patient any number of times with an automatically con-

14. Reger, S. N.: Standardization of Pure Tone Audiometer Testing Technique, Laryngoscope **60**:161 (Feb.) 1950.

trolled, exactly reproducible tone-presentation technique; it is a means of testing any desired number of persons in an invariable manner; it is a technique which a relatively untrained examiner can employ as accurately as a skilled audiometrist; the type of tone presentation makes possible an objective evaluation of the mathematical probability of the accuracy of the patient's response with relatively few data (responses).

Pure-tone audiometry also presents many features which can be standardized. It also makes possible a practical group pure-tone hearing test. A detailed description of the construction of the pure-tone interrupter and the disks employed is given in the paper. Reger also describes the audiometers of Burr and Mortimer and von Békésy which possess desirable self-administering and self-recording features. These also constitute uniform testing procedures. He emphasizes the importance of standardization of manual audiometry and of electromechanically controlled audiometers to introduce more science into the art of audiometry.

Saltzman [15] reports several cases of craniocerebral trauma with perception impairment of hearing in which the recruitment phenomenon was entirely absent.

A new type of speech-hearing test, the Beasley Differential Speech-Hearing Test, was used on a group of 850 patients, of whom 500 had variable degrees of hearing impairments.[16] This test is composed of monosyllabic words of the consonant-vowel-consonant type, which are divided into two lists on the basis of the energy-frequency characteristics of the phonetic elements. The two lists are designated as low-frequency and high-frequency words. A detailed table was set up for all the speech sounds, and a rating scheme was devised for selecting the most differentiating phonetic elements of speech sounds. From this analysis it was concluded that the energy-frequency spectrum is differentiated in relation to the largest number of individual speech sounds in the region of 1500 cycles. On this basis those vowels and consonants were chosen that showed the greatest reduction in articulation when only frequencies above 1500 cycles were eliminated and were influenced the least when frequencies below 1500 cycles were reduced or eliminated. These were classified as high-frequency speech sounds. Likewise, those vowels and consonants showing similar reduction in articulation for frequencies below 1500 cycles and not influenced by the elimination or reduction of those above 1500 cycles were classified as low-frequency speech sounds. The lists of real words were then made up from the respective vowels and consonants in each of the two groups. These have been made into recordings, presenting altogether 12 lists of 10 words each, the words being listed alternately as high-frequency and low-frequency words. A pure-tone audiometer test is first given, followed by the speech audiometer test. The subject listens to the words through the earphones one ear at a time and pronounces the word as heard. The technician then scores the result, striking out the unit or units of each word incorrectly called. The usual procedure is to play the first two tests, high-frequency and low-frequency sounds, at as high a level as 80 db. and then to work up and down in level, depending upon the response of the patient. The objective is to determine the minimum level at which the

15. Saltzman, M.: Recruitment Phenomenon in Craniocerebral Trauma, Arch. Otolaryng. **52**:782 (Nov.) 1950.

16. Beasley, W. C., and Rosenwasser, H.: Determining Factors in Composing and Analyzing Speech-Hearing Tests, Laryngoscope **60**:658 (July) 1950.

patient obtains a 0 score and at least two levels 10 db. apart at which maximum scores are obtained. Between these two end points measurements are made at 10-db. steps and then 5-db. steps as the 0 point is approached.

Data were collected to determine whether the high-frequency and low-frequency articulation could bear any relationship to the pattern of pure-tone audiograms. In the normal group there is no significant difference between the positions and curvature of the low-frequency and high-frequency curves. For those with impaired hearing, the displacement of the low-frequency and high-frequency curves from that of the normal is approximately proportional to the average amount of hearing loss for pure tones at 1000, 1500, and 2000 cycles. In those with different patterns of pure-tone audiograms, the curves are displaced relative to each other in amount and direction proportional to the relative average hearing loss for pure tones below 1000 cycles (low frequency) and above 1000 cycles (high frequency). Prediction, however, of speech-hearing ability from the pattern of pure-tone audiograms is not valid for individual cases, since about 28% of the cases did not conform. This test, however, has a practical value to the otologist, especially in evaluating end results after the fenestration operation. It also has a direct application to the selection of hearing aids in individual cases and appraisal of the nature of the improvement accomplished by different hearing-aid fittings.

Silverman [17] presents the principles underlying articulation tests and the various tests employed, with their clinical application. Articulation-test scores like other test scores are subject to the influence of many variables, requiring conditions to be as constant as possible in making the tests. There are three thresholds of hearing: the threshold of detectability, where sounds are barely detected half of the time; the threshold of perceptibility, where some words are perceived but connected speech cannot be followed, and the threshold of intelligibility, where at least half of the material is understood. For clinical purposes the threshold of intelligibility is the only one needed. The various tests mentioned are the one using the two lists of 42 dissyllabic words of the spondee stress pattern developed at the Psycho-Acoustic Laboratory, of Harvard University, and the one described by Falconer and Davis in which the subject listens to a sample of recorded connected discourse and manipulates the attenuator until he is just able to get the gist of what is being said. His hearing loss is the number of decibels more required by his ear than by the normal ear to reach the threshold of intelligibility for connected discourse. To test auditory discrimination, the phonetically balanced lists are used. A test utilizing 20 equivalent phonetically balanced lists of 50 words each has been constructed by Egan and his associates at the Psycho-Acoustic Laboratory.

Articulation tests are applied clinically in problems of deafness, prognosis, and evaluation of medical and surgical procedure and hearing aids. They are valuable in assessing cochlear function. This applies particularly to the patient who is a candidate for the fenestration procedure. To evaluate therapeutic procedures and hearing aids properly, one should know to what extent the patient's ability to cope with everyday conditions of oral communication has improved. This can be determined by obtaining the social adequacy index for hearing, as introduced by Walsh

17. Silverman, S. R.: Use of Speech Tests for Evaluation of Clinical Procedures, Arch. Otolaryng. **51**:786 (June) 1950.

and Silverman. This index is the average percentage of phonetically balanced words that would be correctly understood by the patient at the faint, the conversational, and the loud speech levels. Articulation testing is still in the stage of experimentation. The methods need to be increasingly refined, validated, and further streamlined for wider clinical application. They are not intended to supplant orthodox clinical tests but are a valuable supplement and should occupy a respectable place in the armamentarium of the clinical otologist.

Bone-conduction audiometry is considered a relatively uncertain method by many because of the variability in technique of applying the bone-conductor vibrator, the lack of clearly established standards among audiometer manufacturers, inadequate testing conditions, and improper masking. Carhart [18] feels that these difficulties can be overcome, and, with future research and greater clinical application, bone-conduction audiometry can be an important diagnostic tool in otology. By using an oscillator of the hearing-aid type held in a steel headband, variabilities caused by positioning and pressure changes can be overcome. The testing room, if not soundproof, should be such that it is adequate for air-conduction testing of normal hearing. This will be adequate for testing bone conduction of patients whose hearing loss is sufficient to constitute a social handicap. The audiometer should be an approved instrument which is functioning satisfactorily. Carhart presents a simple method for the examiner to calibrate the bone-conduction system of his own audiometer. This requires testing 10 to 12 normal-hearing young adults by air conduction and using these average figures as a norm for each frequency. Then 10 to 12 persons with a bilateral, approximately equal, perceptive loss are tested by both air and bone conduction. The average of both curves is plotted, and the amount of each frequency by which the two curves fail to agree represents the correction which must be applied to bone readings for that frequency. Mild room noise does not affect this method of calibration. Finally, one should have an effective system for controlled masking of the opposite ear. When adequate bone-conduction audiometry is done, the results obtained are very useful in diagnosis. Lierle and Reger several years ago stated the basic premise: With bone conduction normal or increased and air-conduction acuity below normal, the patient has a middle-ear lesion; if the patient has the same loss for both air and bone conduction, or possibly a greater decrease for bone-conducted sound, he has an inner-ear lesion; finally, if the patient has a loss for both air- and bone-conducted sound, with the air loss greater than the bone loss, the lesion is a mixed one due to a combined middle- and inner-ear involvement.

Saltzman and Ersner [19] show that shadow hearing by bone conduction cannot be completely eliminated with proper masking. Four cases of total unilateral deafness, with normal air and bone acuity in the other ear, were studied by air conduction. In each was encountered an auditory sensation of an elevated threshold when the bone receiver was applied to the deaf ear while a complex masking sound was placed at the external auditory meatus of the hearing ear. These findings were

18. Carhart, R.: Clinical Application of Bone Conduction Audiometry, Arch. Otolaryng. **51**:798 (June) 1950.

19. Saltzman, M., and Ersner, M. S.: Masking and Shadow Hearing in Bone Conduction, Arch. Otolaryng. **51**:809 (June) 1950.

substantiated when tests under similar conditions were carried out on 10 young adults. The elevation of threshold due to masking is considered essentially a feature of partial or relative refractoriness.

Woods [20] reports his observations on bone conduction investigated in a consecutive series of 39 patients with otosclerosis before and after the fenestration operation. He found the bone conduction showed an average improvement of 11.4 db. Ninety per cent of the patients showed improvement of masked bone conduction after the operation. The same tests were made on the untreated ear for a control. Woods feels that at least part of the bone-conduction loss in otosclerosis is reversible and cannot be attributed to atrophy of disuse or secondary nerve change. In early cases of otosclerosis, air-conduction loss is in the low tones and the first bone-conduction loss shows itself in the high tones. Both of these are reversible by operation. In the later cases neither air- nor bone-conduction losses are wholly reversible. The postoperative audiogram resembles that of an earlier stage of the disease. Woods believes that stapes fixation alone causes a loss in bone conduction and that otosclerosis from the onset produces a mixed deafness, the conductive element being predominant in the early stages and the perceptive element becoming more important as the disease progresses. The bone-conduction tests were made with a Western Electric 2B audiometer and the D-80904 bone-conduction receiver, which was held in the examiner's hand. He regards the bone-conduction receiver as a mechanical tuning fork. The repeat tests when used this way give results quite consistent and comparable with the results of air-conduction audiometry.

MacDonald [21] contends that the mastoid air cells are sound-absorbing elements. To prove this, he has devised an ingenious apparatus in which the bone-conductor head is held in a malleable frame that is held in turn by a steel spring maintaining the apparatus in contact with the patient's head. A shank is attached to the bone-conductor tip which carries an adjustable screw that has a central drill hole large enough to admit a 20-gage nylon bristle. This conducts the vibrations from the bone-conductor's head to the parts being tested. The nylon bristle has a beaded tip made by heating the end over an alcohol flame. After preliminary local anesthesia to the drum membrane, the nylon bristle tip is attached to the short process of the malleus with DuPont cement. The screw attachment is then adjusted so that the patient experiences only slight pressure in the ear. An audiogram is then made with 256, 512, 1024, 2048, and 4096 d. v. Results of 10 consecutive tests on 10 patients are given. This ossicle-conduction test gives a much lower threshold of hearing than does the regular bone-conduction test. The sclerotic mastoid does not absorb sound vibrations as well as the pneumatic mastoid. The mastoid process, however, cannot be compared with ordinary porous material. This test is an office procedure, but it is time-consuming. It may be of value in marginal cases when fenestration is considered, also in those cases in which nerve-deafness has been diagnosed because of poor bone conduction which might be due simply to absorption by the air cells to an extent greater than expected. MacDonald plans to carry out this investigation for all patients with deafness. Up to now it has been done on those with manifest fixation of the stapes selected for fenestration.

20. Woods, R. R.: Bone Conduction in Otosclerosis, Arch. Otolaryng. **51**:485 (April) 1950.

21. MacDonald, P. G.: Bone Conduction Through the Auditory Ossicle Chain, Arch. Otolaryng. **51**:641 (May) 1950.

Campbell [22] brings to our attention the important application of the impedance formula to problems of conduction deafness. This formula, utilized by physicists and engineers for a number of years, essentially states that factors which increase the stiffness of any of the elements of the conduction system will produce a hearing loss predominantly in the lower frequencies, whereas those factors which increase the mass of the elements of the conduction system will produce a hearing loss predominantly in the high-tone range. Examples of increased stiffness are fixation of the footplate of the stapes, adhesions between the ossicles, and contraction of the intra-aural musculature. Increase in mass is found in the presence of pus, blood, or serous fluid in the tympanic cavity. The application of this formula is of importance in studying the progress of many of these conditions. Early fixation of the stapes footplate can be diagnosed at times before an actual hearing loss occurs in members of otosclerotic families by the tilt of the audiogram showing a loss for the low tones and normal hearing for the high. More attention should be given this formula by otologists. It is, however, applicable only to conduction impairments.

Lempert and associates,[23] after three years of investigating the possibility of utilizing electrical responses of the human cochlea in the diagnosis of diseases of the ear, conclude that the recording of these potentials is not a practical clinical procedure. It can be done during a surgical operation, at which time, of course, the clinical decisions have already been made.

The Western Electric 4CA phonograph audiometer, which has served for many years in schools in this country, was used by Bauzá and Martínez [24] for testing hearing among schoolchildren in Uruguay. The authors are impressed by this instrument, which they consider by far the most practical for this purpose, although they realize its shortcomings in the matter of supplying all the information needed for a complete diagnosis. The usual technique and methods prescribed for the instrument were employed. The authors stress the necessity for repeating the examination, particularly in young children, in order to verify the original diagnosis and to judge the effects of treatment.

ACOUSTIC TRAUMA

The effect of jet-engine noise on the cochlear response of the guinea pig was studied by Alexander and Githler.[25] Noise levels of 140 db. and more, with a frequency spectrum of 80 to 8000 cycles, were used, and the extent of recovery in one to three weeks after exposure was shown. The damaged ears were remarkably susceptible to additional trauma which would be safe for normal ears. Further degeneration occurred three to six weeks after stimulation.

Jet-engine noise presents a serious hazard. A plea is made for ear protection and audiometric tests at regular intervals.

---

22. Campbell, P. A.: The Importance of the Impedance Formula in the Interpretation of Audiograms, Tr. Am. Acad. Ophth. **54**:245 (Jan.-Feb.) 1950.

23. Lempert, J.; Meltzer, P. E.; Wever, E. G., and Lawrence, M.: The Cochleogram and Its Clinical Application, Arch. Otolaryng. **51**:307 (March) 1950.

24. Bauzá, C. A., and Martínez, A.: Phonographic Vocal Audiometry in School Children, Arch. pediat. Uruguay **20**:526 (Aug.) 1949.

25. Alexander, I. E., and Githler, F. J.: The Effects of Jet Engine Noise on the Cochlear Response of the Guinea Pig, J. Comp. & Physiol. Psychol. **42**:517 (Dec.) 1949.

In the interesting experiments reported by Huizing,[26] the threshold for a test tone was shifted after exposure to a fatiguing tone. If balance tests are made in persons with perception deafness, a basin-shaped audiogram becomes a horizontal line when intensity is increased. If a patient with perception deafness is exposed to a fatiguing tone of 2000 cps at 30-db. level for three minutes, the shift is greater than normal, because 30 db. above threshold gives relatively greater loudness for a perception-deaf ear than for a normal ear, and the fatigue is greater. This was experimentally proved. In the conditions mentioned above, a threshold shift of up to 12 db. may be considered normal. The greater values produced in hearing aids explain some of the difficulties experienced with them.

Experimental evidence of trauma induced in guinea pigs' hearing apparatus by the noise of pistol shots is described by Krejci and Bornschein.[27] After exposure to the noise of pistol shots, the guinea pigs showed a dip in the electrocochleogram between 3500 and 4500 cps. On vital fixation after 48 hr., the external hair cells and Deiters' cells at the end of the first turn to the middle of the second whorl showed degeneration; ganglion cells, nerve fibers, and middle ear were intact. If the vital fixation was done 17 days after the trauma, only an epithelial sheet was seen in the above-described area, with further degeneration beyond it and retrograde degeneration of the ganglion cells and nerve fibers. Two guinea pigs exposed to the noise of five instead of seven shots showed the same audiometric dip without marked histological changes. Hence, functional impairment alone is possible.

Certain persons are more susceptible to auditory fatigue than others. Wilson [28] suggests the employment of the following tests to demonstrate this prolonged auditory fatigue which should be considered abnormal. The hearing threshold at 4096 cycles is determined for each ear, readings being recorded at 5-db. intervals. After this threshold determination, one ear is exposed to a 2048-cycle fatiguing tone at air intensity of 80 db. for a five-minute period. Immediately after this, the threshold for that ear at 4096 cycles is taken and recorded at ½-min. intervals up to 2 min., then at 1-min. intervals up to 5 min., and, finally, at 8-min. intervals or even longer, until the threshold difference is less than 10 db., which is considered within experimental error. The opposite ear is then given the same test. Two hundred and fifty subjects with normal hearing, whose average age was 26 yr., were given this test. It is known that acoustic fatigue is a protective mechanism for decreasing the sensitivity of the ear during prolonged loud sounds. Abnormality, therefore, is reached only when a prolonged recovery time is encountered. No definite line could be drawn separating the normal from the abnormal. Therefore, a borderline or intermediate group was formed. One hundred and seventy-three subjects recovered from fatigue in less than 2 min. This was considered normal. Thirty-seven subjects required up to 3 min. for recovery and were considered slow but not alarmingly abnormal. Finally, the 40 subjects who showed auditory fatigue of 3 min. or longer were considered definitely abnormal in their delayed recovery.

26. Huizing, H. C.: The Relation Between Auditory Fatigue and Recruitment, Acta oto-laryng., supp. 78, p. 169, 1949.

27. Krejci, F., and Bornschein, H.: Effect of Acoustic Trauma in Guinea Pig: Bioelectric and Histologic Studies, Pract. oto-rhino-laryng. **12**:1, 1950.

28. Wilson, W. H.: Determination of Susceptibility to Abnormal Auditory Fatigue, Ann. Otol. Rhin. & Laryng. **59**:399 (June) 1950.

Schucknecht [29] reports 17 cases of deafness following head injury. The audiograms of many of these patients demonstrate a hearing loss which is severest in the region of the 4096 frequency. The deafness that follows a longitudinal fracture of the temporal bone is of conduction type caused by direct injury of the middle-ear structures which is partially or completely reversible. The transverse fracture of the temporal bone results in a severe nerve-type deafness, with loss of vestibular function in that ear which is usually permanent. Impaired hearing may follow trauma to the head without demonstrable fracture of the temporal bone. The hearing loss is of the nerve type and severest for the high tones, particularly 4096 db. In cases of severe injury all frequencies may be involved. This type of deafness is frequently seen in the ear on the side opposite a fracture of the temporal bone.

Saltzman and Ersner [30] found in studying more than 50 patients who had undergone the fenestration operation that the fenestrated ear could not tolerate an intensity of 100 db. for the middle frequencies. In many cases the ear operated on experienced discomfort at the 80-db. level, whereas the untreated ear was unaffected at 100-db. intensity. These observations seem to justify the conclusion that the altered physiological state of the fenestrated ear produces a definite lowering of the threshold of feeling. No provision has been made in the fenestrated ear to safeguard the cochlea against acoustic trauma. Deterioration of acuity for high tones seen months and years after fenestration operation may be due to acoustic trauma. A practical point is to advise those having a fenestration procedure to protect that ear when exposed to loud sounds, especially when employed in a noisy occupation.

An article on the effects of noise in certain Italian industries is contributed by Mancioli.[31] The author observes that the harmful effects of noise upon the acoustic apparatus have been known for many years. A considerable number of experimental and clinical studies in this field by Gradenigo, Steffannini, D'Onofrio, Beck, Peyser, Opitz, and others, dealing with various occupations, are reviewed. The well-known histological studies by Wittmaack, Popoff, and Malan are cited.

The author's personal observations concern a group of 262 workers engaged in metal-buffing. This occupation involves very loud noise from pneumatic hammers and other sources, as well as considerable bodily vibration imparted from the walls and floors of the factories. Examinations were made by means of an audiometer constructed in Turin, with a range of 32 to 16384 d. v., possessing octave intervals and a field of 130 db. (at 1024 and 2048 d. v.) to 30 db. (at 32 d. v.); both air- and bone-conduction receivers were used. In addition to the otolaryngologic study, general physical and clinical examinations were performed to exclude all possible systemic and local causes of hearing impairment other than occupational factors. Conduction, inner ear, and mixed deafness were all found as apparently resulting from the men's occupation. Representative case histories and audiograms are given in the paper. Tables show the percentages of men with normal hearing, as well as

29. Schucknecht, H. F.: A Clinical Study of Auditory Damage Following Blows to the Head, Ann. Otol. Rhin. & Laryng. **59**:330 (June) 1950.

30. Saltzman, M., and Ersner, M. S.: Threshold of Feeling in the Fenestrated Ear, Arch. Otolaryng. **51**:667 (May) 1950.

31. Mancioli, G.: Contribution to the Study of Industrial Deafness: Audiometric Researches on Metal Buffers, Rass. med. indust. **18**:233 (Nov.-Dec.) 1949.

low-tone, high-tone, and mixed impairments, in different age groups from the second to the fifth decades. No studies were made among older workers for obvious reasons. Tables also present the percentages of men with normal hearing and different types of hearing impairments, based on the length of employment; these are given in five-year periods. The author found that in some cases the static labyrinth was also affected, leading to attacks of vertigo.

Mancioli concludes that acoustic trauma leads primarily to high-tone deafness, which is induced by both air and bone conduction; a possible additional factor is the vibration induced by the machinery. The inhalation of the powder and dust prevalent in the factories also induces congestion of the upper respiratory tract, with catarrhal otitis media complicating and aggravating the inner-ear involvement as a result.

Noise-induced hearing loss was studied by Wheeler.[32] He substantiates many of the fundamental facts already known. Noises the spectrums of which contain a high proportion of high-frequency energy affect the ear more seriously than do noises which are predominantly of low frequency, even though the latter may be at higher intensity levels. The loss tends to occur most extensively in the higher frequencies of the audiogram, the region of maximum loss lying between 2000 and 8000 cycles. When the ear is exposed long enough to noises that are loud enough, the loss may spread to the lower frequencies as the higher-frequency losses become greater. In general, the loss seems maximum at a level one-half to one octave above the stimulus frequency. The recovery factor is variable. Wheeler in his experiments shows that the course of recovery is not a linear function of elapsed time. The recovery rate is very rapid at first and then tends to slow up. Wheeler feels that there is a definite need for predictive or premonitory tests for workmen who are to work in a noisy environment. This view is based on the assumption that certain persons are more vulnerable to noise than others and that tests would enable one to pick out these susceptible persons before their exposure to industrial noise. Such tests would have to be simple, not too time-consuming, and standardized. The other alternative is to retest the employee after a given time of exposure to the actual noisy environment with a given recovery period intervening.

REHABILITATION

Goodhill [33] discusses the importance of the Rh-factor as an etiological factor of a central variety of nerve deafness. The clinical association of moderate bilateral perceptive deafness, athetoid cerebral palsy, and a history of erythroblastosis has been observed. Eight case histories are presented as typical of this group. Kernicterus, the severe central neuropathological complication of erythroblastosis fetalis, is the cause of the athetosis in this group and is probably related to the deafness as well. The deafness is bilateral, and the audiograms are symmetrical for both frequency and intensity. Existing evidence points to the pathological foci being located in the nuclear masses of the brain and brain stem. The incidence of erythroblastosis in a large sampling of perceptively deafened children appears to be

32. Wheeler, D. E.: Noise-Induced Hearing Loss, Arch. Otolaryng. **51**:344 (March) 1950.

33. Goodhill, V.: Nuclear Deafness and the Nerve Deaf Child: The Importance of the Rh Factor, Tr. Am. Acad. Ophth. **54**:671 (July-Aug.) 1950.

significantly higher than would be expected in the general population. The Rh-factor, as a result of these findings, begins to play a role as a potential cause of some cases of infantile "nerve-deafness."

Barnett and Ryder [34] report a case of kernicterus associated with hemorrhagic disease of the newborn. The infant, now 14 mo. of age, has pronounced brain damage evidenced by deafness and profound general retardation.

Guild [35] reports in considerable detail four cases of children who either developed while under observation, or showed initially, a marked degree of progression of an abrupt type of high-tone loss. In all four instances the impairments progressed gradually. The exact cause of this condition is as yet unknown. There is a possibility, however, that a follow-up study of a large number of persons from childhood until middle adult life might provide some basic information on the commonest type of hearing impairment that occurs in man. Such a study would also furnish information on and a better understanding of the clinical prognosis of this condition.

Williams and his co-workers [36] report a series of cases of impairment of hearing due to endolymphatic hydrops without vertigo. The criteria necessary for a tentative diagnosis of such a condition are the finding of inner-ear deafness with a tendency toward a greater loss of hearing for low tones than for the high tones, which is even more apparent on the bone-conduction curve than on the air-conduction curve of the audiogram. The hearing impairment may be of sudden onset, occurring in two to three days, followed by persistence of the loss. There may be a history of considerable variability in the degree of deafness, and in women it may be increased before, during, and shortly after the menstrual period. There may also be a history of vasomotor rhinitis, urticaria, dermatitis factitia, or severe unilateral headache often associated with increased deafness. Treatment consists of intravenous histamine therapy daily for variable periods until a maximal therapeutic result is apparently secured, followed by prolonged therapy with either histamine phosphate solution or monoethanolamine nicotinate solution ("nicamin"), given subcutaneously by the patient himself. After the prescribed period, if symptoms such as tinnitus return, the patient is instructed to administer therapy again until they are relieved.

Day,[37] in his Wherry Memorial Lecture presented at the 1950 annual session of the American Academy of Ophthalmology and Otolaryngology, gave a comprehensive picture of the present-day management of deafness. He discussed the various types of deafness, their etiological factors, and methods of therapy. He stressed the obligation the otologist has to his hard-of-hearing patients. Those patients who cannot be benefited by any form of therapy and have irreversible hearing loss should be given proper advice. Their problems should be discussed frankly with them and an explanation given them of the nature of their deafness

34. Barnett, R. N., and Ryder, C. F.: Deafness and Kernicterus, Arch. Otolaryng. **52**:771 (Nov.) 1950.

35. Guild, S. R.: The Progression of Impaired Hearing for High Tones During Childhood, Laryngoscope **60**:885 (Sept.) 1950.

36. Williams, H. L.; Horton, B. T., and Day, L. A.: Endolymphatic Hydrops Without Vertigo, Arch. Otolaryng. **51**:557 (April) 1950.

37. Day, K. M.: The Management of Deafness, Tr. Am. Acad. Ophth. **55**:22 (Sept.-Oct.) 1950.

and why they cannot be helped. The otologist should gain their confidence, be positive and firm in his advice, and give them encouragement, not sympathy. The otologist should never tell a patient he will become totally deaf or that his hearing will get worse. Day stated:

> It takes time and effort to convince hard of hearing people that they must accept their handicap as an actuality before they can be guided onto the path leading to necessary readjustments and rehabilitation. If we consider ourselves to be real otologists we will gladly expend this time and effort in their behalf.

Conservation of the hearing function, according to Lempert,[38] can be accomplished in three ways: first, by avoiding damage to the normally functioning hearing organ; secondly, by improving the hearing function when it is impaired and conserving the improved hearing, and, finally, by preventing or delaying further impairment of function when improvement is impossible. Methods of preventing middle-ear infections in childhood are given, and the proper judicious treatment for these infections is stressed. Lempert emphasizes the importance of utilizing proper, thorough operative procedures in treatment of chronic suppurative ear infections and of conserving as much hearing as possible by avoiding the radical procedures when a more conservative thorough operation will eradicate the disease but still conserve the hearing. The fenestration operation plays a very important part in treatment of that group of cases in which hearing impairment already exists. It should also be considered as a possibility of conserving hearing and allaying secondary nerve changes. Lempert also brings up the question whether the amplified sound over the entire hearing range as produced by the electric hearing aid can overstimulate the ear and damage cochlear function. He believes that use of hearing aids can produce acoustic trauma in middle-ear deafness, since they also amplify the frequencies which are relatively unimpaired; thus, when hearing aids are used over a long period, the sound intensity can injure the hair cells of Corti. This possibility remains until hearing aids are built so that the otologist will be in a position to prescribe the necessary degree of intensity to which each pure-tone frequency should be adjusted so as to amplify no more than necessary to meet the functional needs of each frequency.

Fowler[39] discusses the various diseases and conditions responsible for hearing impairments and stresses the utilization and employment of adequate therapeutic measures to prevent the otitic complications or to cure them rapidly to avoid hearing defects. This applies to the various contagious and infectious diseases. He also thoroughly investigates all children in otosclerotic families to try to find early evidences of and clues to the disease so as to get as much information as possible to aid in eventual detection and, possibly, proper treatment of these patients in the future. Prevention is of primary importance at all levels of hearing loss.

Lewis[40] reports the progress made with the expanded program carried out for the rehabilitation of the preschool deaf child at the Massachusetts Eye and Ear Infirmary. The effectiveness of this program is based upon proper diagnosis,

38. Lempert, J.: Conservation of Hearing Function, Tr. Am. Acad. Ophth. **54**:713 (July-Aug.) 1950.

39. Fowler, E. P.: Prevention of Deafness, Arch. Otolaryng. **51**:42 (Jan.) 1950.

40. Lewis, D. K.: Rehabilitation of the Preschool Deaf Child, Laryngoscope **60**:564 (June) 1950.

estimation of the hearing capacity, parental education, early application of remedial measures, and a continuous and sympathetic follow-up program. The diagnosis must consider other conditions which cause a failure of language development. These include motor speech delay, developmental retardation, lack of motivation, and emotional traumas. Each of these conditions has significant characteristics and must be carefully considered in the diagnosis of deafness in the very young child. The importance of parental education in the program is emphasized. The estimation of residual hearing requires more than the simple reflex responses and audiogram. A careful history from the parent is necessary, concerning what the child hears and in what circumstances and whether he makes attempts at vocalization. The child's reflex response to sound stimuli should be observed. The learned and conditioned auditory response is very important. This can be done by having the child take his turn at a simple game, doing his part at the command of a sound. This can be demonstrated to the parents, who can then carry it out further at home. Observation of the child's basic language ability gives valuable information. A psychometric evaluation is desirable, if facilities are available. The vestibular tests and the galvanic skin-resistance tests give added information under certain conditions.

The remedial measures consist of a program of acoustic training, speech reading, and language development. If the residual hearing is amenable to re-education and the child's psychometric evaluation is favorable, he is given a hearing aid. The use of the hearing aid, especially for the profoundly deaf child, must be followed by expert guidance in speech therapy and auditory training. Of a group of 55 children trained by the clinic, 43 are able to attend public or private schools and only 12 are in a school for the deaf. Eight of 17 profoundly deaf children are in public schools. Another group investigated elsewhere showed that only 47 of 134 children are in the public or private schools and 87 are in the schools for the deaf. Only two of the 75 profoundly deaf children are in the public schools. Lewis mentions the lack of facilities in this country for proper rehabilitation of the preschool deaf child. The majority of these children can be rehabilitated through early intensive remedial measures to the level of public-school placement. The cooperation, enthusiasm, and efforts of the otologist are necessary for the development of such programs.

Langsam[41] stresses the importance of hearing re-education as an important adjunct in therapy and rehabilitation, especially of those patients with a hearing loss of 40 db. or less not due to a stapes fixation. It is a valuable help to those patients with a high-frequency loss who are wearing hearing aids. The aid only amplifies the sound, but re-education interprets the sound to the patient's brain. It has proved satisfactory in cases of boilermaker's disease, in which there is a precipitous drop in hearing for the high-frequency sounds. The technique consists of specially planned exercises taught to the patient and to his relative or friend, who can carry them out as home exercises or treatments. The exercises are built up from simple to difficult ones. A course of treatment may last from six weeks to six months. The improvement in hearing will be noticed by those with whom the

41. Langsam, W. L.: Hearing Re-Education: Its Applicability, Eye, Ear, Nose & Throat Month. **39**:556 (Oct.) 1950.

patient comes in contact and will not be evident on the audiogram, as he has not been trained to recognize the pure-tone audiometer sounds. It will also be evident in tests using word survey lists.

Morrissett,[42] in a complete critical review of the subject of auditory nerve-deafness, shows the ineffectiveness and uselessness of all present therapy for this condition. He stresses the importance of aural rehabilitation as the only practical method of management presently available for these patients. He discusses the various phases of such a program as has been carried out by the Armed Forces during and since World War II. These patients require essentially a complete medical and otological study, complete audiometric and auricular ability tests, psychologic study, provision and proper utilization of a hearing aid, instruction in lip reading, auditory training, voice training and speech correction, speech insurance, if indicated, and vocational guidance, if indicated. Obviously such a program requiring expensive equipment, specially trained personnel, and adequate facilities is beyond the realm of the individual otologist. It can be managed only by certain hospitals or hearing or speech centers in each community. Where such organizations are not available, one should consider the possibility of a number of otologists grouping together to establish and utilize such a clinic for the proper management of all cases of impaired hearing, especially the auditory-nerve type.

42. Morrissett, L. E.: Plight of the Nerve-Deaf Patient, Arch. Otolaryng. **51**:1 (Jan.) 1950.

Abstracts from Current Literature

Ear

OTOGENOUS EDEMA OF THE BRAIN. HANS BRUNNER, Ann. Otol., Rhin. & Laryng. **58**:997 (Dec.) 1949.

Otogenous edema of the brain is supposed to present the clinical symptoms of otogenous brain abscess. This fact would imply that otogenous brain edema has no symptomatology of its own. The diagnosis rarely is made prior to operation and was not made in the two cases presented by Brunner. This is attributable to the comparatively infrequent occurrence of otogenous edema of the brain and the desire of the surgeon not to overlook an eventual brain abscess. However, the correct diagnosis can be made, although the differential diagnosis between brain abscess and serous meningitis of inflammatory or hypertensive type and brain edema may be difficult.

In this respect, the author considers the following findings of importance in cases of otogenous edema of the brain: 1. Brain edema occurs most frequently in children and young adults. 2. The patient is not acutely ill; there is no dehydration and no prostration. 3. At the onset there usually is fever and leucocytosis. Chills eventually occur. 4. Fluctuating headache may be present. Drowsiness and slow cerebration do not occur, except in children following a protracted course of the illness, or in patients suffering from epileptic attacks. Fluctuation of the intensity of drowsiness in these instances has been reported. 5. There may be venous engorgement of the retina, but there is no marked degree of papilledema. 6. The spinal fluid pressure is normal or is slightly raised. There may be a slight increase in cells and albumin. However, in cases of hypertensive serous meningitis, increase of spinal fluid pressure up to 950 mm., with an average increase of more than 300 mm., has been reported. 7. Encephalography fails to show changes in the ventricles. 8. "Irritative" brain symptoms—Jacksonian attacks, cerebellar fits, opisthotonos, lateral deviation of the eyes, twitching of the facial nerve, involuntary movements of the body, rotation of the body, and involuntary deglutition—are common at the onset of the disease. Among 21 cases of serous inflammation of the brain and leptomeninges reported in the literature (including the two cases presented and another of the author's cases previously reported), "irritative" symptoms were noted in 11 cases. These symptoms when present at the onset of the disease are of great importance in diagnosis. 9. Other neurological symptoms occurring at the onset of otogenous edema of the brain are monoplegia, hemiplegia, facial paralysis, sensory aphasia, cerebellar symptoms, and eventual hemianopsia. 10. While otogenous edema of the brain appears to occur most frequently in acute infections of the temporal bone, there are cases on record in which brain edema occurred in chronic otitis or subsequent to radical mastoid operations. 11. At operation, the dura is normal or pachymeningitis is present. After incision of the dura, the leptomeninges appear to be normal unless there is an associated serous meningitis. The cerebral cortex is slightly hyperemic, and there is a moderate herniation of the brain subsequent to the incision of the dura. There is no discoloration of the cortex prior to a puncture. Puncture of the brain does not meet with the sense of resistance encountered when an encapsulated abscess is present and does not release fluid of any kind, unless the cannula enters the inferior horn of the ventricle. The brain herniation recedes rapidly after decompression.

Brunner states that it may be less difficult to establish the differential diagnosis between brain edema and brain abscess than to distinguish between brain edema and serous meningitis. In the latter case, a distinction must be established between different localizations of the same disease and not between two different morbid entities.

Treatment of otogenous edema of the brain attempts removal of the focus of infection and decompression of the brain. Mastoid surgery is employed in removal of the focus of infection. The dura should be exposed in all instances. So far as decompression is concerned, the condition of the patient should determine whether immediate surgery or conservative measures should be applied.

STEELE, Kansas City, Kan.

RADIUM THERAPY FOR LYMPHOID TISSUE IN THE NASOPHARYNX. EDWIN B. BILCHICK, and ALBERT R. KOLAR, Ann. Otol., Rhin. & Laryng. **59**:78 (March) 1950.

The authors report findings on 282 patients of all ages treated by nasopharyngeal irradiation with the 50 mg. radium applicator. Of 120 patients with conductive deafness, 73, or 61%, showed improvement (gain of more than 10 db. for frequencies 500, 1,000 and 2,000 averaged together). In the 2 to 15 year age group, 77% of the patients showed improvement, while in the 15 to 75 age group 50% were improved.

While irradiation has no beneficial effect on patients with nerve deafness, there may be some improvement in the conductive loss of hearing in patients with mixed deafness. The destructive effects of irradiation of lymphoid tissue and the proliferative effects on epithelium, with its possible dangers, are described. Emphasis is placed upon cautious selection of cases and technique of application. Special danger may be incurred by repeated series of treatments.

STEELE, Kansas City, Kan.

EVALUATION OF IRRADIATION OF PHARYNGEAL AND NASOPHARYNGEAL LYMPHOID TISSUE. FRANCIS L. LEDERER, Ann. Otol., Rhin. & Laryng. **59**:102 (March) 1950.

When the available past and present evidence is considered, irradiation of the nasopharynx by the standardized techniques seemingly is without danger. However, this form of therapy should be used with restraint because of our incomplete knowledge of radiobiology and the empiricism of the present dosage. Emphasis should be placed upon the importance of recognizing the risk from irradiation to the physician and his staff.

The use of nasopharyngeal irradiation is not a substitue for surgery and should be confined to cases of conductive deafness associated with lymphoid hyperplasia in and about the orifices of the Eustachian tube. Transitory hearing loss, allergic manifestations, and infections in the nose, sinuses, or nasopharynx must be recognized and treated by the otolaryngologist, in addition to irradiation. The use of irradiation in patients having irreverisble hearing loss is to be deplored and discouraged.

Careful history-taking in relation to previous irradiation is of importance because of the possibility that a repeated series of treatments may produce untoward tissue changes.

Lederer states that if competent administration is assumed no great advantage is to be had from radium over roentgen irradiation in the treatment of Eustachian lymphoid hyperplasia. The simplicity of obtaining and using the nasopharyngeal radium applicator eventually may lead to avoidable harm.

STEELE, Kansas City, Kan.

ON THE TREATMENT OF OTOGENIC BRAIN ABSCESSES. J. A. HARPMAN, J. Laryng. & Otol. **64**:319 (June) 1950.

This is an excellent article based on the author's experience in the treatment of 18 patients with otogenic brain abscess. Harpman feels that otogenic brain abscesses should be operated on as soon as diagnosed and that it is advisable for one to do the required aural surgery before operating on the brain. He advocates the endaural approach and describes his technique in detail; he believes that labyrinthotomy should also be done when the labyrinth is nonfunctioning. According to the author, temporal lobe abscesses are better drained through a mastoidectomy; his exposure for this is obtained by extending the endaural incision upwards. He removes the mastoid and middle ear tegmen and a large area of squama. The dura is disinfected and incised and the brain explored with a blunt instrument such as a bayonet forceps. When the abscess cavity is entered, pus will escape alongside the exploring instrument. When an abscess is found, the opening is enlarged and its contents thoroughly evacuated. The cavity is packed with gauze impregnated with iodoform and mineral oil.

Cerebellar abscesses of otogenic origin are best drained through the mastoid if Trautmann's triangle is large enough. Often a preoperative dural fistula occurs in this area, and cerebellar herniation is less likely to occur here than by the separate approach. Initial complete evacuation of the abscess is of great importance. Several other methods of treatment of brain abscess are

discussed. Harpman is opposed to excision of brain abscesses of otogenic origin because of the greater possibility of more permanent brain damage than that resulting from more conservative treatment.

The author stresses the use of penicillin and other antibiotics both parenterally and intrathecally and of the sulfonamides in the treatment of such lesions. The diagnostic aid of air and electroencephalograms and of angiography and ventriculography is discussed.

Of the first eight patients, seven died. Of the last 10 patients, only one died and nine recovered. This decrease in mortality is attributed to early surgical treatment, thorough initial evacuation of the abscess, and use of sulfonamides and antibiotics.

LE JEUNE and LEWIS, New Orleans.

AUREOMYCIN IN TREATMENT OF OTITIC AND OPHTHALMIC HERPES ZOSTER. EDWARD W. GANS, Laryngoscope **60**:939 (Sept.) 1950.

Gans has treated herpes zoster with aureomycin with good results. The amount of aureomycin used was 500 mg. every six hours for adults, just half that used by Finland in his series. In addition, vitamin B, physical therapy, and iodides were used. Six cases were presented.

HITSCHLER, Philadelphia.

OFFICE PROCEDURE IN HEARING EVALUATION: A PRACTICAL APPROACH. LEROY L. SAWYER, Laryngoscope **60**:1061 (Nov.) 1950.

Sawyer discusses which tests are reliable and practical from the standpoint of equipment and time consumption for office evaluation of hearing.

There must be a testing room whose ambient noise level is not greater than 45 db. Reliable tests can then be performed with the use of the pure tone audiometer and of the recorded voice. Details of these tests are discussed.

Davis' Social Adequacy Index was applied. Its use was considered most valuable in determining the hearing loss for speech.

HITSCHLER, Philadelphia.

AURAL MANIFESTATION OF SCURVY. JOHN L. BURGESS, Texas J. Med. **18**:283 (Aug.) 1947.

Burgess, in discussing the aural manifestations of scurvy, describes the hemorrhagic blebs frequently seen in the external auditory canal and on the drum membrane following upper respiratory infections. He refers to an article published in 1938 by Ruskin, who was struck by the similarity in the descriptions of the hemorrhagic lesions found in infantile scurvy and of the lesions in the external ear following influenza. Ruskin was of the opinion that the otological picture was the first manifestation of scurvy and reported 10 cases of the conditions in which he had had excellent results with calcium ascorbate. Burgess reports 22 cases observed by him since 1938 in which large doses of vitamin C and calcium gluconate were used with gratifying results. Burgess summarizies his articles as follows:

"1. The so-called otitis of influenza and myringitis bullosa hemorrhagica are otological manifestations of scurvy.

"2. Systemic treatment for vitamin C deficiency should be instituted."

SINGLETON, Dallas, Tex.

BLAST RUPTURE OF THE TYMPANIC MEMBRANE. THOMAS W. FOLBRE, Texas J. Med. **43**:397 (Oct.) 1947.

Folbre reports 55 cases of tympanic membrane perforations resulting from blast injury from high explosives. The patients were seen in the European Theater of Operations during the years 1944 and 1945. The patients were seen from four days to three weeks following the injury and remained under his observation for an average period of 48 days. There seemed to be no definite relation between rupture of the drum membrane and the degree of impaired hearing.

In some, there was no permanent impairment of the hearing; in others the hearing impairment was marked. When present, the hearing impairment was usually of the mixed type. In 27 patients there was middle ear infection. Two required mastoid surgery; the mastoid disease was exudative, with little evidence of bone destruction. Treatment of the infected ruptures consisted of intramuscular injections of penicillin, dry cleansing of the ears, and dusting of small amounts of sulfanilamide powder into the perforation. The smaller perforations which did not close were treated by placing a small sterile piece of cigaret paper over the perforations and touching the paper with a small cotton-tipped applicator saturated with trichloroacetic acid. The patches were left in place from two to four weeks according to the size of the perforation. Eleven perforations out of a total of 15 treated by the above method closed. The author states that his method can be utilized on certain patients in civilian practice. SINGLETON, Dallas, Tex.

SURGICAL TREATMENT OF OTOSCLEROSIS. J. MIONDONSKI and H. GANS. Acta otol-laryng. **38**:159 (April) 1950.

In the otolaryngological clinic of Cracow, Poland, 144 patients with otosclerosis were subjected to the fenestration operation in the period of September, 1946, to October, 1949. Of this group, 100 have been under observation for periods up to three years. The operation was performed by a retroauricular technique and done under a combination of basal anesthesia and local procaine anesthesia. A Leitz 10.5 binocular microscope was used. In addition to audiometric and tuning fork threshold tests, observation "water curves" were made in a series of patients.

The authors believe that the type of postoperative nystagmus that occurs is an index of the delicacy of the surgery. If the labyrinth is not injured, the immediate postoperative nystagmus is directed toward the treated side. If the immediate postoperative nystagmus is directed toward the side not operated on, it indicates that the labyrinth was not adequately protected from harm. Statistically in a group of 100 patients, including five patients on whom reoperations were performed, the percentage of gain in hearing of a useful degree was 81.

In some cases a copper preparation was administered intravenously to prevent osteogenesis. The improvement in hearing has been maintained in this group, but the group is not claimed to be a statistical sample.

LEWY, Chicago.

Nose

RHINOPLASTY: ITS SURGICAL COMPLICATIONS AND HOW TO AVOID THEM. I. B. GOLDMAN, J. Internat. Coll. Surgeons, **13**:283 (March) 1950.

In Goldman's paper the familiar complications incidental to rhinoplastic surgery are presented and discussed. The subject is covered adequately and in excellent detail, but it can be summed up only as a review of the content of many papers which have already appeared, without adding anything new to the subject of rhinoplasty. The many illustrations are superior in meaning and quality.

SELTZER, Philadelphia.

THE MANAGEMENT OF NASOPHARYNGEAL FIBROMAS. FREDERICK A. FIGI and ROBERT E. DAVIS, Laryngoscope **60**:794 (Aug.) 1950.

Nasopharyngeal fibromas are limited to the period of adolescence and most of them regress spontaneously by the age of 20. They are highly vascular, locally invasive, and destructive in nature. They occur almost entirely in males. Fibrous tumors resembling juvenile fibromas occasionally appear at any age but are innocuous and therefore must be differeniated.

Juvenile fibromas are easily recognized clinically, and, because of the possibility of profuse uncontrollable bleeding, biopsy is often better omitted. Formerly, treatment of these tumors was mainly surgical, but, because of hemorrhage and recurrence, radiation, and more recently a combination of interstitial radiation and surgical diathermy, has been used successfully. Also, the approach to the tumor was formerly intranasal and/or intraoral. Recently the transantral

approach has proved to be of great value. Occasionally, ligations of the external carotid artery, on one side or both, are performed because of hemorrhage at operation or preoperatively. The latter is, however, not usually deemed necessary.

Many other important data are presented in this comprehensive paper.

HITSCHLER, Philadelphia.

CONGENITAL CHOANAL ATRESIA. FRANCIS H. MCGOVERN, Laryngoscope **60**:815 (Aug.) 1950.

Congenital choanal atresia is discussed with special reference to the clinical, physiological, and developmental aspects.

Five new cases are reported and the literature reviewed. Many interesting findings are mentioned. There may very likely be a hereditary factor present. Development of the sinuses is usually normal, there is normal ciliary action, and biopsy of the nasal membranes shows no abnormality. The hearing is usually likewise normal.

The condition may be dangerous in infants owing to possible suffocation, but there is no danger and little discomfort in adults.

HITSCHLER, Philadelphia.

RHINOLOGY IN CHILDREN: RÉSUMÉ AND COMMENTS ON THE LITERATURE FOR 1949. D. E. S. WISHART, Laryngoscope **60**:849 (Sept.) 1950.

As always, Wishart's résumé of rhinology in children is excellent. Almost every subject is touched upon: the common cold, sinusitis, allergy, tonsils, radium treatment, and others. The article should be read in full.

HITSCHLER, Philadelphia.

PRIMARY TUMORS OF THE NASAL SEPTUM. F. A. SOOY, Laryngoscope **60**:964 (Oct.) 1950.

Sooy reviews the literature on tumors of the nasal septum and adds 11 cases of his own. Each type of tumor is discussed separately. Methods of diagnosis, including cytological diagnosis, and treatment for each lesion are included.

HITSCHLER, Philadelphia.

EPISTAXIS—CASE REPORT: REPORT OF A CASE OF LIGATION OF THE EXTERNAL CAROTID AND ANTERIOR ARTERIES, AND SECTION OF THE POSTERIOR ETHMOIDAL ARTERY. HARRY SACKADORF, Laryngoscope **60**:1125 (Nov.) 1950.

This case illustrates the fact that it is the ethmoidal arteries which supply the nasal cavity above the middle turbinate. When uncontrollable hemorrhage occurs in this region, these vessels must be ligated. This procedure is preferable to ligation of the internal carotid artery.

The area below the middle turbinate is supplied by the external carotid.

HITSCHLER, Philadelphia.

MIXED TUMOR OF SALIVARY GLAND TYPE IN THE NOSE. SAMUEL BAER, and CARTER M. ALEXANDER, N. Y. J. Med. **50**:2206 (Sept.) 1950.

This case report comes from the department of otolaryngology and pathology, Samaritan Hospital. A Negro woman, aged 28 yr., was examined on March 25, 1949, for obstruction of the right nostril. She complained of epistaxis and epiphora on the right, and pain in the right cheek. There was no loss of weight or other symptoms or signs. Rhinoscopy showed a swollen, bluish mass, which resembled hypertrophy of the inferior turbinate, occluding the right nostril. It was stony hard, bled easily and profusely, and did not shrink under drugs. Transillumination and roentgenograms of the sinuses disclosed nothing significant. Results of blood studies and tests for syphilis were also negative. After blocking of the right sphenopalatine ganglion, removal of the growth was carried out. The hardness proved to be due to a mere shell within which was thick caseous material. Laboratory diagnosis was "mixed tumor of salivary gland type." Nine months after operation, no recurrence was found. There was no difficulty in breathing, epiphora had ceased and the patient was back at work as a stenographer. A figure showing the epithelial nests containing cystic glands in a mucoid and partly cartilaginous stroma accompanies the report.

VOORHEES, New York.

Larynx

TRACHEOTOMY IN BOTULISM: REPORT OF A CASE. BENJAMIN BOFENKAMP, and ROBERT E. PRIEST, Ann. Otol., Rhin. & Laryng. **58**:1093 (Dec.) 1949.

Bofenkamp and Priest report a case in which botulism was treated by tracheotomy in addition to the conventional methods of treatment.

Other investigators have shown that botulinus toxin acts peripherally rather than directly upon the central nervous system, the essential action being that of paralysis of the motor nerve end plates of the voluntary nervous system and a blocking of the motor and secretory fibers of the parasympathetic portion of the autonomic nervous system. Presence of the toxin decreases the synthesis of acetylcholine, which action may cause the neuroparalytic phenomena involving the parasympathetic nervous system. The nerves of special sense, the sympathetic division of the autonomic system, and the sensory fibers of the peripheral nerves are unaffected by the toxin.

Study of the case histories appearing in the literature impressed upon Bofenkamp and Priest the similarity of respiratory distress due to pharyngeal pooling of mucus in cases of bulbar poliomyelitis and botulism. Since tracheotomy has relieved respiratory distress in properly selected cases of bulbar poliomyelitis, it was decided to utilize it in the case at hand. Additional treatment included the administration of botulinus antitoxin and penicillin, artificial respiration by mechanical respirator, and frequent pharyngeal and tracheal suction for removal of secretions. After 16 days, the tracheotomy tube was removed and the patient was comfortable when out of the respirator. About three weeks later he left the hospital.

STEELE, Kansas City, Kan.

RESECTION AND ANASTOMOSIS OF THE TRACHEA: AN EXPERIMENTAL STUDY. JOHN H. GRINDLAY, O. THERON CLAGETT and HERMAN J. MOERSCH, Ann. Otol., Rhin. & Laryng. **58**:1225 (Dec.) 1949.

This report describes the result 18 to 28 yr. after resection of a portion of trachea and anastomosis over a polyethylene (polythene®) tube in a series of seven dogs. Grindlay, Clagett, and Moersch reported previously early data on the present series of dogs (*Arch. Surg.* **57**:253-266 [Aug.] 1948), observations at that time having covered periods of two and one half to 13 mo.

The technique of tracheal resection and anastomosis over a polyethylene tube is described.

Bronchoscopy has shown that the implanted tube does not seem to cause obstruction to the outflow of bronchial secretions, as was feared originally. Absence of cough seems to support the belief that outflow of mucus is not impeded significantly, despite the fact that there is an interruption of continuity of the ciliated epithelium of the trachea. Presumably, air movement during respiration maintains a balance between the amounts of mucus at each end of the tube. The tube does not become coated with mucus because polyethylene is not wetted by aqueous solutions and will not cohere to other substances. Tubes in situ do not appear to deteriorate and do not seem to act as irritants.

Two dogs have been observed for more than two years and five dogs for 18 mo. after operation. Bronchoscopic examination revealed few signs that the tubes are injurious to the animals, except for one dog in which the tube was too large and one in which it was too small in diameter. In the former, there was an area of partial tracheal collapse distal to the tube, and in the latter, the tube fit loosely. One dog coughed out its tube about a year after operation, presumably because the tube loosened because it was too small. However, bronchoscopy showed only a slight irregularity at the site of anastomosis. The necessity for accurate fitting of the tube at the time of tracheal resection is illustrated thereby.

Basing their reasoning upon their period of observation, the authors conclude that resection and anastomosis of the trachea may have some clinical application. A primary tracheal tumor, localized to a few segments, conceivably might be treated surgically by this method.

STEELE, Kansas City, Kan.

TUMORS OF THE LARYNX. OLIVER W. SUEHS, Texas J. Med. **43**:393-407 (Oct.) 1947.

Suehs, in discussing tumors of the larynx, states that they are rare in infants and young children. Multiple papilloma is the commonest laryngeal tumor found in children; it produces dyspnea in most cases. Removal with cupped forceps or electrocoagulation without injury to the vocal cords is the treatment of choice. These tumors show a marked tendency to recur until the age of puberty.

Vocal nodules are an occasional cause of hoarseness in children; they are often bilateral and are caused by vocal abuse.

In adults, chronic inflammation plus vocal abuse are the primary factors in the origin of most of the benign tumors. Polyps, single papilloma, vocal nodules, and diffuse inflammatory thickenings are the lesions most frequently encountered in the order named. Hoarseness is the chief symptom, but obstruction may sometimes occur. Treatment is directed toward restoration of a normal voice; if obstruction is present, it must be cared for.

Of the malignant tumors, sarcoma is rare. Squamous cell carcinoma is the lesion most frequently encountered. The increasing use of tobacco and alcohol, vocal abuse, and heredity are suspected of being factors in the cause of laryngeal cancer. Hoarseness is an early symptom of cordal cancer. If the lesion is remote from the cords, hoarseness is a late symptom. Pain and dyspnea often occur as late symptoms. Pain, when preesnt, is often referred to the ear on the side of the greater involvement. Disturbance in swallowing may be the first symptoms of a lesion at the upper orifice of the larynx. Diagnosis is made on the history of the case, a careful indirect and direct examination of the larynx, and a histologic examination of a representative piece of removed tissue. It is sometimes necessary to repeat the biopsy. Cancer of the larynx must be differentiated from tuberculosis, syphilis, and certain benign neoplasms.

In the treatment of laryngeal cancer one may choose bewteen surgery and irradiation or may combine the two. Lesions confined to the cords are best treated by surgical removal done with the laryngofissure technique. More extensive lesions may be treated by laryngectomy in selected cases. If the lesion is inoperable, irradiation is the treatment of choice.

SINGLETON, Dallas, Tex.

Directory of Otolaryngological Societies *

INTERNATIONAL

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

Meeting: Third Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology
Place: Habana, Cuba. Time: January, 1952.
President: Dr. José Gros. Information may be secured from Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

SECOND LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY

President: Prof. A. de Paula Santos.
Secretaries: Dr. José de Rezende Barbosa, Dr. Plinio de Mattos Barretto, Hospital das Clinicas, São Paulo, Brazil.
Place: São Paulo, Brazil. Time: July, 1951.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianopolis.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: Chicago. Time: June 11-13, 1952.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Derrick Vail, 700 N. Michigan Avenue, Chicago 11.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn
Place: Palmer House, Chicago. Time: Oct. 14-20, 1951.

AMERICAN BOARD OF OTOLARYNGOLOGY

Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Palmer House, Chicago. Time: Oct. 9-12, 1951.
Place: Royal York Hotel, Toronto, Canada. Time: May 13-16, 1952.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Herman J. Moersch, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. H. Marshall Taylor, 111 W. Main St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 128 So. 10th St., Philadelphia 2.
Place: Royal York Hotel, Toronto, Canada. Time: May 23-24, 1952.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Royal York Hotel, Toronto, Canada. Time: May 20-22, 1952.

SECTIONS:

Eastern.—Chairman: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Southern.—Chairman: Dr. Lester A. Brown, 490 Peachtree St., Atlanta.
Middle.—Chairman: Dr. Ralph J. McQuiston, 20 N. Meridian St., Indianapolis.
Western.—Chairman: Dr. Howard P. House, 1136 W. 6th St., Los Angeles.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Royal York Hotel, Toronto, Canada. Time: May 18-19, 1952.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

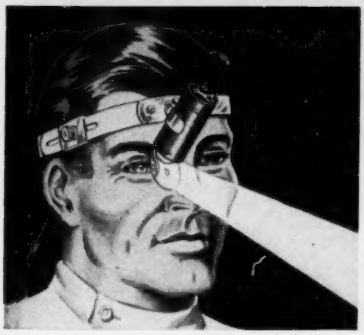
President: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.
Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. William H. Evans, 24 Wick Ave., Youngstown 3, Ohio.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

MECHANICAL PERFECTION TO AID SURGICAL SKILL



Perfect illumination for all body cavities: throat, nose, ear, larynx, oral surgery. dental surgery, brain surgery, eye surgery, gynecology, bronchoscopy, fenestration, proctoscopy.

GOOD-LITE

THE PHYSICIANS UNIVERSAL HEADLIGHT

Constructed with finest scientific craftsmanship to give you an instrument of enduring quality . . .

Good-Lite is one of the finest, most brilliant headlights ever constructed. You have 750 foot candles directed exactly where you want it and to a depth you desire. Good-Lite is completely adjustable so that it will fit all doctors. It may be used over one eye for deep, narrow cavities, or between the eyes for shallow cavity work, or at the side for oblique illumination. May be readily adjusted for parallel, divergent or convergent rays. Removal of mirror permits use as direct headlight.

GOOD-LITE UNIT INCLUDES

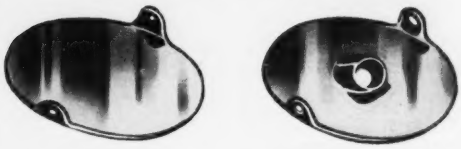
Good-Lite Parallel Headlight
Two mirrors
Extra long life bulbs
Variable 15 watt transformer
Molded shock-proof rubber connections
White acetate plastic headband, leather or fiber, (form-fitting)

Sold at leading medical and dental supply houses— or use coupon.

GOOD-LITE CO.
7638 MADISON STREET
FOREST PARK, ILLINOIS

COMPLETE WITH TWO MIRRORS

. . . Twin Mirrors Are Readily Interchangeable.



PLAIN MIRROR **PEEP HOLE MIRROR**

1. PLAIN MIRROR—For shallow cavities such as throat, eye, vagina, fenestration and other surface work.
2. PEEP HOLE MIRROR—Center of beam light for deep cavities such as nose, larynx, ear, bronchoscopy. Has protective shield, open aperture—no glass to cloud or blur.
3. DIRECT LIGHT for surface surgery by simply removing mirror.

GOOD-LITE CO., DEPT. O
7638 MADISON STREET, FOREST PARK, ILLINOIS

☐ Please send me illustrated booklet describing in detail the Good-Lite Parallel Ray Headlight.

☐ Please send me_____Good-Lite Parallel Ray Headlight units, complete with extra long life bulb, variable 15 watt transformer, molded shock-proof connections. @ $32.50 ea.

☐ White acetate plastic, ☐ leather, or ☐ fiber form-fitting headband

Name_____

Address_____

City_____ Zone_____ State_____

AND NOW *A New Grip on Problems of Industrial Medicine*

THROUGH A Journal Designed For Your Interests

A. M. A. Archives of INDUSTRIAL HYGIENE and OCCUPATIONAL MEDICINE

Edited by men of outstanding reputation in the fields of industrial health and preventive industrial hygiene: Prof. Philip Drinker, Boston, Chief Editor; Robert Kehoe, M.D., Cincinnati; James Sterner, M.D., Rochester, N. Y.; Frank Patty, Detroit, Mich.; Theodore Hatch, Pittsburgh; Frank Princi, M.D., Cincinnati; Fenn E. Poole, M.D., Glendale, Calif.; William A. Sawyer, Rochester, N. Y.

Covering the research and field aspects of industrial hygiene and the clinical and medical aspects of occupational industrial health programs.

Merging the best features of *Occupational Medicine* and *The Journal of Industrial Hygiene and Toxicology*.

Integrated closely with the activities of the Council of Industrial Health of the A. M. A. and the American Industrial Hygiene Association.

A Better and More Useful Journal of Industrial Medicine! Such is the result which the Editorial Board of A. M. A. Archives of INDUSTRIAL HYGIENE and OCCUPATIONAL MEDICINE has achieved by combining parallel publications.

To the thousands of physicians who are directly or indirectly concerned today with medicine in industry, the new journal will bring reports of the continuing and important developments in the field, with original articles covering problems and day to day experiences of physicians in industry; an excellent abstracting service similar to that carried in the *Journal of Industrial Hygiene and Toxicology;* additional foreign journal abstracting; reviews.

Whether servicing industrial firms, attending employees, applying to general practice some of the findings of industrial medicine, or merely *watching* this expanding field, you will want to receive this vital new periodical.

AMERICAN MEDICAL ASSOCIATION
535 N. Dearborn Street, Chicago 10

Enter my subscription to A. M. A. Archives of INDUSTRIAL HYGIENE and OCCUPATIONAL MEDICINE to start *with the next issue.* Per year, $8.00 in U. S. (Canadian, $8.40; Foreign, $9.00).

I enclose check..... Please bill me.....

Name.....

Address.....

storz
Surgical Instruments

A Correct and Approved Model of

Neivert Tonsil Snare

Precisely constructed of Stainless Steel... Each $19.50

This snare has a number of improvements over the Eve snare, but is functionally the same instrument.

a. Wire ends are inserted into the tubular stylet — they are completely enclosed — will not puncture operator's fingers or patient's mouth or lips.

b. The barrel is made of a solid rod, not of half sections, which gives more stability and lets the ring section slide easily.

c. Thumb ring is attached by a cap nut — and the finger ring section stops ¾" from end of shaft when being drawn closed to prevent pinching fingers.

d. The cap nut holds the thumb ring securely but is easily removed and attached. Pliers are not necessary.

Ask for New Ear, Nose, Throat Instrument Catalogue

Storz Instrument Co. 4570 AUDUBON AVE., ST. LOUIS 10, MO.

SPECIAL SERVICES

for the

HARD OF HEARING ADULT

HEARING COUNSELING SERVICE to help your patient understand his hearing problem and to help in the selection of a hearing aid if one is advisable.

LIP READING and AUDITORY TRAINING to help your patient use his remaining hearing efficiently and to help him integrate what he sees with what he hears.

VOICE and SPEECH WORK to help your patient retain pleasing tone quality and clear speech.

THE NITCHIE SCHOOL OF LIP READING, INC.

Founded 1903

342 Madison Avenue New York 17, N. Y.
near 43rd St. MUrray Hill 2-6423
Chartered by the New York State Board of Regents

WHEN A HEARING AID IS NECESSARY

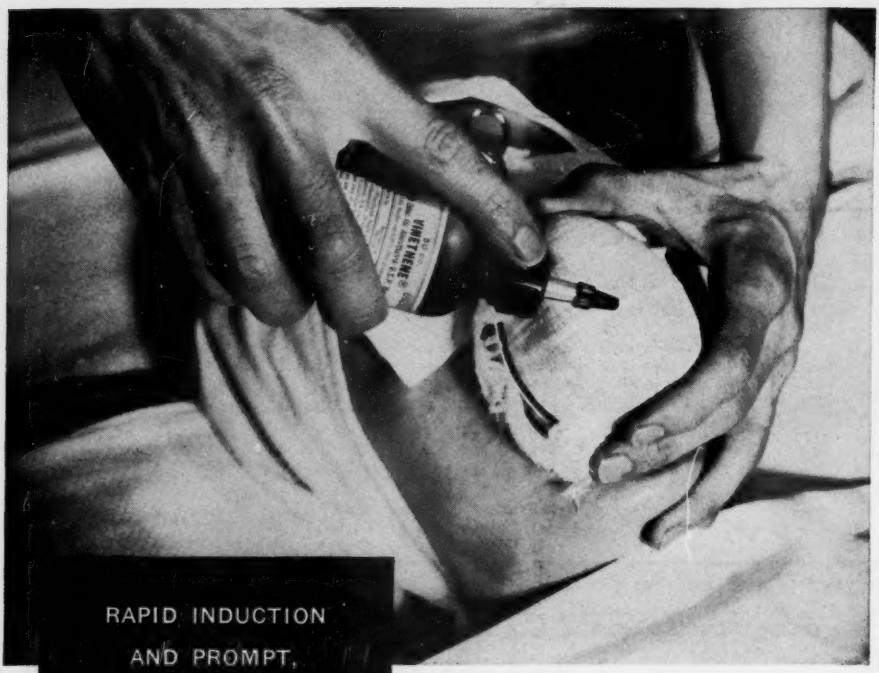
you can rely on PARAVOX to supply ample volume, clearness of tone, and other desirable qualities, all backed by a widespread service plan. The PARAVOX Tiny-MYTE Hearing Aid, shown above, is being worn by thousands with satisfaction.

PARAVOX Hearing Aids are widely known in the medical profession and were recently on exhibit at the San Francisco Annual Session of the AMA, the Annual Meetings of the American Academy of Ophthalmology and Otolaryngology, the Medical Society of Penna., the Mississippi Valley Medical Society.

Accepted by the Council on Physical Medicine and Rehabilitation, American Medical Association

PARAVOX, INC.

2056 East 4th St., Cleveland 15, Ohio



RAPID INDUCTION
AND PROMPT,
UNEVENTFUL RECOVERY
IN

Anesthesia of SHORT DURATION

Reduced emotional hazards through rapid induction, and minimal after-effects may be achieved in short procedures often encountered in daily practice with the use of Vinethene, a practical inhalation anesthetic. In addition to its use in such procedures as changing of painful dressings, reduction of fractures, myringotomy, abscess incisions, Vinethene also is recommended for induction prior to ether anesthesia, and for complementing nitrous oxide-oxygen.

Literature on request.

VINETHENE®

(Vinyl Ether for Anesthesia U. S. P. Merck)

AN INHALATION ANESTHETIC FOR SHORT OPERATIVE PROCEDURES

COUNCIL ACCEPTED

MERCK & CO., Inc.
Manufacturing Chemists
RAHWAY, NEW JERSEY
In Canada: MERCK & CO. Limited—Montreal

facilities unlimited

. . . at your disposal

The Imperatori Treatment and Diagnostic Unit places no limitations on your inherent skill in ear, nose and throat work. It's modern—complete—efficient—attractive.

For instance, there's a large electric panel for cautery and transillumination; a unique antrum wash bottle; a rubber-mounted, spring-suspended Motor-compressor Unit which is noiseless and vibrationless. The pump compartment is reinforced and ventilated to prevent overheating, and the cautery handle is the latest pistol-grip type with permanently connected insulated electric cord and two standard nasal or laryngeal cautery tips.

The heavy gauge steel cabinet (with all joints welded) is finished in chipless white enamel . . . working top and turret top are of formica . . . acid, burn and stain-proof. (Optional cabinet finish in grained walnut, grained mahogany, or special color at little additional cost.)

Illustrated brochure on request.

Sklar

LONG ISLAND CITY, N. Y.

A SKLAR PRODUCT

IMPERATORI
Treatment and Diagnostic unit

Available through accredited surgical supply distributors

A Preferred Nasal Decongestant and Vasoconstrictor

Otorhinolaryngologists frequently express preference for Neo-Synephrine hydrochloride to alleviate turgescence and nasal congestion in colds, sinusitis and various forms of rhinitis.

"When considerable nasal obstruction exists, relief may be obtained by the instillation of some shrinking agent into the nose . . . as for example Neo-Synephrine hydrochloride (¼%)"[1]

A "desirable preparation of this type has been perfected in Neo-Synephrine hydrochloride. It may be used for local application in the nose in ¼ to 1% solution."[2]

Neo-Synephrine's "desired effect occurs within from two to fifteen minutes . . ."[3]

"Its action is sustained for two hours or more."[3]

Neo-Synephrine hydrochloride is notable for freedom from sting and for effectiveness on repeated application. There are few complaints of after effects such as burning and nasal congestion . . . and little tendency to develop local sensitivity.[1]

NEO-SYNEPHRINE® HYDROCHLORIDE

Prompt and Prolonged Relief in

COLDS • SINUSITIS • HAY FEVER

Winthrop-Stearns INC.

NEW YORK 18, N. Y. WINDSOR, ONT.

1. Tuft, L.: Clinical Allergy. Philadelphia, W. B. Saunders Co., 1947, pp. 335-336.
2. Hansel, F. K.: Allergy of the Nose and Paranasal Sinuses. St. Louis, C. V. Mosby Co., 1936, p. 769.
3. Kelley, S. F.: Choice of Sympathomimetic Amines. Cornell Conferences on Therapy, II, 1947, p. 156.

Neo-Synephrine, trademark reg. U. S. & Canada, brand of phenylephrine